МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ

УЧРЕЖДЕНИЕ ОБРАЗОВАНИЯ
«ГОМЕЛЬСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ»

Кафедра военной и экстремальной медицины

К. М. СЕМУТЕНКО, А. А. РЕБКО,
Д. А. ПРОКОПОВИЧ

МЕДИЦИНА ЭКСТРЕМАЛЬНЫХ СИТУАЦИЙ

Учебно-методическое пособие
для студентов 4 курса факультета по подготовке специалистов
для зарубежных стран медицинских вузов

В двух частях

Часть 2
ТОКСИКОЛОГИЯ ЭКСТРЕМАЛЬНЫХ СИТУАЦИЙ

MEDICINE OF EXTREME SITUATIONS

Teaching workbook
for 4th year students of the Faculty of preparation of experts
for foreign countries of medical higher educational institutions

In two parts

Part 2
TOXICOLOGY OF EXTREME SITUATIONS

Гомель
ГомГМУ
2017

УДК 355.233/.237 (07)
ББК 68.9я7
С 74

Рецензенты:

доктор медицинских наук, полковник медицинской службы,
начальник кафедры военно-полевой терапии,
профессор военно-медицинского факультета
Белорусского государственного медицинского университета
А. С. Рудой;

кандидат медицинских наук, доцент, подполковник медицинской службы,
начальник кафедры военной и экстремальной медицины
Гродненского государственного медицинского университета
В. А. Новоселецкий

Семутенко, К. М.

С 74 Медицина экстремальных ситуаций: учеб.-метод. пособие для студентов 4 курса факультета по подготовке специалистов для зарубежных стран медицинских вузов: в 2 ч. = Medicine of extreme situations.: Teaching workbook for 4th year students of the Faculty of preparation of experts for foreign countries of medical higher educational institutions: in two parts. — P. 2: Toxicology of extreme situations / К. М. Семутенко, А. А. Ребко, Д. А. Прокопович. — Гомель: ГомГМУ, 2017. — Ч. 2: Токсикология экстремальных ситуаций. — 192 с.

ISBN 978-985-506-921-2

В учебно-методическом пособии представлен материал по токсикологической характеристике, патогенезу действия, клинической картине, диагностике и лечению отравлений некоторыми отравляющими веществами, широко распространенными в различных отраслях промышленности и народного хозяйства.

Предназначено для студентов 4 курса факультета по подготовке специалистов для зарубежных стран медицинских вузов.

Утверждено и рекомендовано к изданию научно-методическим советом учреждения образования «Гомельский государственный медицинский университет» 29 декабря 2016 г., протокол № 6.

УДК 355.233/.237 (07)
ББК 68.9я7

ISBN 978-985-506-920-2
ISBN 978-985-506-921-2 (ч. 2)

CONTENTS

Introduction 4
Chapter 1. Introduction to Toxicology 5
Chapter 2. Nuclear Explosion 14
Chapter 3. Chemical detection technologies 40
Chapter 4. Nerve agents 55
Chapter 5. Mustard (blister) agents 85
Chapter 6. Lung Damaging Agents (Choking Agents) 99
Chapter 7. Blood Agents 106
Chapter 8. Toxic Alcohols 137
Chapter 9. Vomiting Agents 154
Chapter 10. Incapacitating Agents 162
Chapter 11. Biotoxins 169

INTRODUCTION

The purpose of this Guide is to provide a basic reference for students, health and medical professionals in disaster medicine. It is intended for use in education, training and planning by all health personnel.

In this context, the terminology «health and medical personnel» is used to include personnel providing first aid, ambulance, medical doctor, nursing, mental health, public health, scientific and other associated expertise and involvement.

Disasters come in many shapes and forms, and physicians stand at the front line to help detect, support, direct, and participate during all disasters that might strike a community. Thus, it is critical that any disaster medicine curriculum encompass a full spectrum of disaster types and responses.

Members of the local health care system are among the first to respond when a disaster strikes. Every physician has the potential to be in a situation where he or she may need to respond. Knowledge of how to respond to disasters and how to coordinate that response with other agencies and organizations involved is essential.

Some disasters can result in the destruction of a considerable portion of the community's medical resources and take a considerable toll on life. Traditional principles of disaster medicine that come into play during an event include preparatory drills involving hospital systems, triage, evacuation, local and country response, public health, vector management, and personal safety. During the recovery phase, continuity of operations must be addressed to sustain the business of health care. This document must be written into health care disaster plans in order to support hospital systems and clinics in the path of disaster. It is important for physicians to consider this part of the recovery phase because of the critical nature of their practices to the communities they serve.

Last time there has been an increased focus on the standardization of disaster response in an effort to speed the implementation of relief and decrease the iatrogenic component of the chaos inherent to such situations.

Effective planning, coordination, and execution are keys to successful disaster response. Debriefings capture lessons learned and facilitate strategic plan revisions. When tailored to the needs of the individual, psychological debriefings performed by trained health care professionals allow healing for both victims and responders.

Disasters might occur on or near national land. While the focus of the specific objectives in this guide is domestic disasters, the majority of the principles covered may also be applied to disaster responses abroad.

CHAPTER 1
INTRODUCTION TO TOXICOLOGY

Toxicology can be defined as that branch of science that deals with poisons, and a poison can be defined as any substance that causes a harmful effect when administered, either by accident or by design, to a living organism. By convention, toxicology also includes the study of harmful effects caused by physical phenomena, such as radiation of various kinds, noise, and so on. In practice, however, many complications exist beyond these simple definitions, both in bringing more precise definition to the meaning of poison and to the measurement of toxic effects. Broader definitions of toxicology, such as «the study of the detection, occurrence, properties, effects, and regulation of toxic substances», although more descriptive, do not resolve the difficulties. Toxicity itself can rarely, if ever, be defined as a single molecular event, but is, rather, a cascade of events starting with exposure, proceeding through distribution and metabolism, and ending with interaction with cellular macromolecules (usually DNA or protein) and the expression of a toxic end point. This sequence may be mitigated by excretion and repair. It is to the complications, and to the science behind them and their resolution, that this textbook is dedicated, particular to the *how* and *why* certain substances cause disruptions in biologic systems that result in toxic effects. Taken together, these difficulties and their resolution circumscribe the perimeter of the science of toxicology.

The study of toxicology serves society in many ways, not only to protect humans and the environment from the deleterious effects of toxicants, but also to facilitate the development of more selective toxicants such as anticancer and other clinical drugs, pesticides, and so forth.

Poison is a quantitative concept, almost any substance being harmful at some doses but, at the same time, being without harmful effect at some lower dose. Between these two limits, there is a range of possible effects, from subtle long-term chronic toxicity to immediate lethality. Vinyl chloride may be taken as an example. It is a potent hepatotoxicant at high doses, a carcinogen with a long latent period at lower doses, and apparently without effect at very low doses. Clinical drugs are even more poignant examples because, although therapeutic and highly beneficial at some doses, they are not without deleterious side effects and may be lethal at higher doses. Aspirin (acetylsalicylic acid), for example, is a relatively safe drug at recommended doses and is taken by millions of people worldwide. At the same time, chronic use can cause deleterious effects on the gastric mucosa, and it is fatal at a dose of about 0.2–0.5 g/kg. Approximately 15 % of reported accidental deaths from poisoning in children result from ingestion of salicylates, particularly aspirin.

The importance of dose is well illustrated by metals that are essential in the diet but are toxic at higher doses. Thus, iron, copper, magnesium, cobalt, man-

ganese, and zinc can be present in the diet at too low a level (deficiency), at an appropriate level (maintenance), or at too high a level (toxic). The question of dose-response relationships is fundamental to toxicology.

The definition of a poison, or toxicant, also involves a qualitative biological aspect because a compound, toxic to one species or genetic strain, may be relatively harmless to another. For example, carbon tetrachloride, a potent hepatotoxicant in many species, is relatively harmless to the chicken. Certain strains of rabbit can eat *Belladonna* with impunity while others cannot. Compounds may be toxic under some circumstances but not others or, perhaps, toxic in combination with another compound but nontoxic alone. The methylenedioxyphenyl insecticide synergists, such as piperonyl butoxide, are of low toxicity to both insects and mammals when administered alone, but are, by virtue of their ability to inhibit xenobiotic-metabolizing enzymes, capable of causing dramatic increases in the toxicity of other compounds.

The measurement of toxicity is also complex. Toxicity may be acute or chronic, and may vary from one organ to another as well as with age, genetics, gender, diet, physiological condition, or the health status of the organism. As opposed to experimental animals, which are highly inbred, genetic variation is a most important factor in human toxicity since the human population is highly outbred and shows extensive genetic variation. Even the simplest measure of toxicity, the LD_{50} (lethal dose; the dose required to kill 50 % of a population under stated conditions) is highly dependent on the extent to which the above variables are controlled. LD_{50} values, as a result, vary markedly from one laboratory to another.

Exposure of humans and other organisms to toxicants may result from many activities: intentional ingestion, occupational exposure, environmental exposure, as well as accidental and intentional (suicidal or homicidal) poisoning. The toxicity of a particular compound may vary with the portal of entry into the body, whether through the alimentary canal, the lungs, or the skin. Experimental methods of administration such as injection may also give highly variable results; thus, the toxicity from intravenous (IV), intraperitoneal (IP), intramuscular (IM), or subcutaneous (SC) injection of a given compound may be quite different. Thus, toxicity may vary as much as 10-fold with the route of administration. Following exposure, there are multiple possible routes of metabolism, both detoxifying and activating, and multiple possible toxic end points.

Attempts to define the scope of toxicology, including that which follows, must take into account that the various subdisciplines are not mutually exclusive and are frequently interdependent. Due to overlapping of mechanisms as well as use and chemical classes of toxicants, clear division into subjects of equal extent or importance is not possible.

Many specialized terms are used in the various subdisciplines of toxicology as illustrated in the *Dictionary of Toxicology*. However, some terms are of particular importance to toxicology in general; these and some more recent terms are defined in the glossary to be found at the end of this volume.

Although B through F (following) include subdivisions that encompass essentially all of the many aspects of toxicology, there are two new approaches (A, following) that serve to integrate the discipline as a whole.

A. Integrative Approaches

1. *Bioinformatics*. In the narrow and original meaning, bioinformatics was the application of information technology to molecular biology. While this is still the most important aspect of bioinformatics, it is increasingly applied to other fields of biology, including molecular and other aspects of toxicology. It is characterized by computationally intensive methodology and includes the design of large databases and the development of techniques for their manipulation, including data mining.

2. *Systems Biology.* Although systems biology has been defined in a number of ways, some involving quite simple approaches to limited problems, in the currently most commonly accepted sense, it is an integrative approach to biological structure and function that will be of increasing importance to biology in general and toxicology in particular. In large part, biology has been reductionist throughout its history, studying organs as components of organisms, cells as components of organs, enzymes, nucleic acids, and so on, as components of cells, with the goal of describing function at the molecular level. Systems biology, on the other hand, is holistic and has the objective of discerning interactions between components of biological systems and describing these interactions in rigorous mathematical models. Furthermore, the proponents of systems biology aim to integrate these models at higher and higher levels or organization in order to develop an integrated model of the entire organism.

Clearly, systems biology is in its infancy; however, the ultimate value of having an integrative model that could clarify all of the effects, from the most proximate to the ultimate, of a toxicant on a living organism, will provide enormous benefits not only for fundamental studies but in such applied areas as human health risk assessment.

B. Modes of Toxic Action. This includes the consideration, at the fundamental level of organ, cell, and molecular function, of all events leading to toxicity *in vivo*: uptake, distribution, metabolism, mode of action, and excretion. The term mechanism of toxic action is now more generally used to describe an important molecular event in the cascade of events leading from exposure to toxicity, such as the inhibition of acetylcholinesterase in the toxicity of orga-nophosphorus and carbamate insecticides. Important aspects include the following:

1. *Biochemical and molecular toxicology* consider events at the biochemical and molecular levels, including enzymes that metabolize xenobiotics, generation of reactive intermediates, interaction of xenobiotics or their metabolites with macromolecules, gene expression in metabolism and modes of action, signaling pathways in toxic action, and so on.

2. *Behavioral toxicology* deals with the effects of toxicants on animal and human behavior, which is the final integrated expression of nervous function in

the intact animal. This involves both the peripheral and central nervous systems, as well as effects mediated by other organ systems, such as the endocrine glands.

3. *Nutritional toxicology* deals with the effects of diet on the expression of toxicity and with the mechanisms of these effects.

4. *Carcinogenesis* includes the chemical, biochemical, and molecular events that lead to the large number of effects on cell growth collectively known as cancer.

5. *Teratogenesis* includes the chemical, biochemical, and molecular events that lead to deleterious effects on development.

6. *Mutagenesis* is concerned with toxic effects on the genetic material and the inheritance of these effects.

7. *Organ toxicity* considers effects at the level of organ function (e.g., neurotoxicity, hepatotoxicity, and nephrotoxicity).

C. Measurement of Toxicants and Toxicity. These important aspects deal primarily with analytical chemistry, bioassay, and applied mathematics, and are designed to provide the methodology to answer certain critically important questions. Is the substance likely to be toxic? What is its chemical identity? How much of it is present? How can we assay its toxic effect, and what is the minimum level at which this toxic effect can be detected? A number of important fields are included:

1. *Analytical toxicology* is a branch of analytical chemistry concerned with the identification and assay of toxic chemicals and their metabolites in biological and environmental materials.

2. *Genomics.* The sometimes stated distinction that genomics deals with genomes while molecular biology deals with single genes is unrealistic and unnecessary; it is more appropriate to regard genomics as an aspect of molecular biology that deals not only with genomes and gene expression but also such important aspects as genetic polymorphisms, particularly single nucleotide polymorphisms (SNPs). Techniques, such as microarrays, are now available to examine simultaneously the expression of very large numbers of genes.

3. *Proteomics* deals with the protein complement of organisms, the entire complement being known as the proteome. Thus, while genomics is concerned with gene expression, proteomics examines the products of the expressed genes.

4. *Metabolomics* is the next step in the sequence from genomics through proteomics and is concerned with the profile of small molecules produced by the metabolic processes of an organism. Changes in the profile in response to chemical stress are of importance to both fundamental and applied toxicology.

5. *Toxicity testing* involves the use of living systems to estimate toxic effects. It covers the gamut from short-term tests for genotoxicity such as the Ames test and cell culture techniques to the use of intact animals for a variety of tests from acute toxicity to lifetime chronic toxicity. Although the term «bioassay» is used properly only to describe the use of a living organism to quantitate the amount of a particular toxicant present, it is frequently used to describe any *in vivo* toxicity test.

6. *Toxicologicpathology* is that branch of pathology that deals with the effects of toxic agents manifested as changes in subcellular, cellular, tissue, or organ morphology.

7. *Structure-activity* studies are concerned with the relationship between the chemical and physical properties of a chemical and toxicity and, particularly, the use of such relationships as predictors of toxicity.

8. *Biomathematics and statistics* relate to many areas of toxicology. They deal with data analysis, the determination of significance, and the formulation of risk estimates and predictive models.

9. *Epidemiology:* as it applies to toxicology, is of great importance as it deals with the relationship between chemical exposure and human disease in actual populations, rather than in experimental settings.

D. Applied Toxicology. This includes the various aspects of toxicology as they apply in the field or the development of new methodology or new selective toxicants for early application in the field setting.

1. *Clinical toxicology* is the diagnosis and treatment of human poisoning.

2. *Veterinary toxicology* is the diagnosis and treatment of poisoning in animals other than humans, particularly livestock and companion animals, but not excluding feral species. Other important concerns of veterinary toxicology are the possible transmission of toxins to the human population in meat, fish, milk, and other foodstuffs, and the care and ethical treatment of experimental animals.

3. *Forensic toxicology* concerns the medicolegal aspects, including detection of poisons in clinical and other samples.

4. *Environmental toxicology* is concerned with the movement of toxicants and their metabolites and degradation products in the environment and in food chains, and with the effect of such contaminants on individuals and, especially, populations. Because of the large number of industrial chemicals and possibilities for exposure, as well as the mosaic of overlapping laws that govern such exposure, this area of applied toxicology is well developed.

5. *Industrial toxicology* is a specific area of environmental toxicology that deals with the work environment and constitutes a significant part of *industrial hygiene.*

E. Chemical Use Classes. This includes the toxicology aspects of the development of new chemicals for commercial use. In some of these use classes, toxicity, at least to some organisms, is a desirable trait; in others, it is an undesirable side effect. Use classes are not composed entirely of synthetic chemicals; many natural products are isolated and are used for commercial and other purposes and must be subjected to the same toxicity testing as that required for synthetic chemicals. Examples of such natural products include the insecticide, pyrethrin, the clinical drug, digitalis, and the drug of abuse, cocaine.

1. *Agricultural chemicals* include many compounds, such as insecticides, herbicides, fungicides, and rodenticides, in which toxicity to the target organism is a desired quality whereas toxicity to «nontarget species» is to be avoided. De-

velopment of such selectively toxic chemicals is one of the applied roles of comparative toxicology.

2. *Clinical drugs* are properly the province of pharmaceutical chemistry and pharmacology. However, toxic side effects and testing for them clearly fall within the science of toxicology.

3. *Drugs of abuse* are chemicals taken for psychological or other effects and may cause dependence and toxicity. Many of these are illegal but some are of clinical significance when used correctly.

4. *Food additives* are of concern to toxicologists only when they are toxic or being tested for possible toxicity.

5. *Industrial chemicals* are so numerous that testing them for toxicity or controlling exposure to those known to be toxic is a large area of toxicological activity.

6. *Naturally occurring substances* include many phytotoxins, mycotoxins, minerals, and so on, all occurring in the environment. The recently expanded and now extensive use of herbal «remedies» and dietary supplements has become a cause of concern for toxicologists and regulators. Not only is their efficacy frequently dubious, but their potential toxicity is also largely unknown.

7. *Combustion products* are not properly a use class but are a large and important class of toxicants, generated primarily from fuels and other industrial chemicals.

F. Regulatory Toxicology. These aspects, concerned with the formulation of laws, and regulations authorized by laws, are intended to minimize the effect of toxic chemicals on human health and the environment.

1. *Legal aspects* are the formulation of laws and regulations and their enforcement. In the United States, enforcement falls under such government agencies as the Environmental Protection Agency (EPA), the Food and Drug Administration (FDA) and the Occupational Safety and Health Administration (OSHA). Similar government agencies exist in many other countries.

2. *Risk assessment* is the definition of risks, potential risks, and the risk-benefit equations necessary for the regulation of toxic substances. Risk assessment is logically followed by *risk communication* and *risk management.* Risk assessment, risk communication, and risk management are frequently referred to as *risk analysis.*

Relationship to other sciences

Toxicology is a highly eclectic science and human activity drawing from, and contributing to, a broad spectrum of other sciences and human activities. At one end of the spectrum are those sciences that contribute their methods and philosophical concepts to serve the needs of toxicologists, either in research or in the application of toxicology to human affairs. At the other end of the spectrum are those sciences to which toxicology contributes.

In the first group, chemistry, biochemistry, pathology, physiology, epidemiology, immunology, ecology, and biomathematics have long been important while molecular biology has, in the last two or three decades, contributed to

dramatic advances in toxicology.

In the group of sciences to which toxicology contributes significantly are such aspects of medicine as forensic medicine, clinical toxicology, pharmacy, and pharmacology, public health, and industrial hygiene. Toxicology also contributes in an important way to veterinary medicine, and to such aspects of agriculture as the development and safe use of agricultural chemicals. The contributions of toxicology to environmental studies have become increasingly important in recent years.

Clearly, toxicology is preeminently an applied science, dedicated to the enhancement of the quality of life and the protection of the environment. It is also much more. Frequently, the perturbation of normal life processes by toxic chemicals enables us to learn more about the life processes themselves. The use of dinitro-phenol and other uncoupling agents to study oxidative phosphorylation and the use of a-amanitin to study RNA polymerases are but two of many examples. The field of toxicology has expanded enormously in recent decades, both in numbers of toxicologists and in accumulated knowledge. This expansion has brought a change from a primarily descriptive science to one which utilizes an extensive range of methodology to study the mechanisms involved in toxic events.

Brief history of toxicology

Much of the early history of toxicology has been lost, and in much that has survived, toxicology is of almost incidental importance in manuscripts dealing primarily with medicine. Some, however, deal more specifically with toxic action or with the use of poisons for judicial execution, suicide, or political assassination. Regardless of the paucity of the early record, and given the need for people to avoid toxic animals and plants, toxicology must be one of the oldest practical sciences.

The Egyptian papyrus, *Ebers*, dating from about 1500 BC, must rank as the earliest surviving pharmacopeia, and the surviving medical works of Hippocrates, Aristotle, and Theophrastus, published during the period 400–250 BC, all include some mention of poisons. The early Greek poet Nicander treats, in two poetic works, animal toxins (Therica) and antidotes to plant and animal toxins (Alexipharmica). The earliest surviving attempt to classify plants according to their toxic and therapeutic effects is that of Dioscorides, a Greek employed by the Roman emperor Nero about 50 AD.

There appear to have been few advances in either medicine or toxicology between the time of Galen (131–200 AD) and that of Paracelsus (1493–1541). It was the latter who, despite frequent confusion between fact and mysticism, laid the groundwork for the later development of modern toxicology by recognizing the importance of the dose-response relationship. His famous statement «All substances are poisons; there is none that is not a poison. The right dose differentiates a poison and a remedy» succinctly summarizes that concept. His belief

in the value of experimentation was also a break with earlier tradition.

There were some important developments during the eighteenth century. Probably the best known is the publication of Ramazini's *Diseases of Workers* in 1700 which led to his recognition as the father of occupational medicine. The correlation between the occupation of chimney sweeps and scrotal cancer by Percival Pott in 1775 is almost as well-known although it was foreshadowed by Hill's correlation of nasal cancer and snuff use in 1761. Orfila, a Spaniard working at the University of Paris in the early nineteenth century, is generally regarded as the father of modern toxicology. He clearly identified toxicology as a separate science and, in 1815, published the first book devoted exclusively to toxicology. An English translation in 1817 was entitled *A General System of Toxicology or, A Treatise on Poisons, Found in the Mineral, Vegetable and Animal Kingdoms, Considered in Their Relations with Physiology, Pathology and Medical Jurisprudence.* Workers of the late nineteenth century who produced treatises on toxicology include Christian, Kobert, and Lewin. The recognition of the site of action of curare by Claude Bernard (1813-1878) began the modern study of the mechanisms of toxic action. Since then, advances have been numerous — too numerous to list in detail. They have increased our knowledge of the chemistry of poisons, the treatment of poisoning, the analysis of toxicants and toxicity, as well as modes of toxic action and detoxication processes, and specific molecular events in the poisoning process.

With the publication of her controversial book, *The Silent Spring*, in 1962, Rachel Carson became an important influence in initiating the modern era of environmental toxicology. Her book emphasized stopping the widespread, indiscriminate use of pesticides and other chemicals and advocated use patterns based on sound ecology. Although sometimes inaccurate and with arguments often based on frankly anecdotal evidence, her book is often credited as the catalyst leading to the establishment of the U.S. EPA and she is regarded by many as the mother of the environmental movement.

It is clear, however, that since the 1960s, toxicology has entered a phase of rapid development and has changed from a science that was largely descriptive to one in which the importance of mechanisms of toxic action is generally recognized. Since the 1970s, with increased emphasis on the use of the techniques of molecular biology, the pace of change has increased even further, and significant advances have been made in many areas, including chemical carcinogenesis and xenobiotic metabolism, among many others.

Dose-response relationships

As mentioned previously, toxicity is a relative event that depends not only on the toxic properties of the chemical and the dose administered but also on individual and interspecific variation in the metabolic processing of the chemical.

The first recognition of the relationship between the dose of a compound and the response elicited has been attributed to Paracelsus (see Section 1.3). It is noteworthy that his statement includes not only that all substances can be toxic at some dose, but that «the right dose differentiates a poison from a remedy», a concept that is the basis for pharmaceutical therapy.

For many chemicals and effects, there will be a dose below where no effect or response is observed. This is known as the *threshold dose*. This concept is of significance because it implies that a *no observed effect level* (NOEL) can be determined and that this value can be used to determine the safe intake for food additives and contaminants such as pesticides. Although this is generally accepted for most types of chemicals and toxic effects, for chemical carcinogens acting by a genotoxic mechanism, the shape of the curve is controversial, and for regulatory purposes, their effect is assumed to be a no-threshold phenomenon.

Sources of toxic compounds

Given the enormous number of toxicants, it is difficult to classify them, either chemically, by function, or by mode of action since many of them would fall into several classes. Some are natural products, many are synthetic organic chemicals of use to society, while some are byproducts of industrial processes and waste disposal. It is useful, however, to categorize them according to the expected routes of exposure or according to their uses.

A. Exposure Classes. Exposure classes include toxicants in food, air, water, and soil as well as toxicants characteristic of domestic and occupational settings. Toxicant use classes are described in detail in Chapter 3.

B. Use Classes. Use classes include drugs of abuse, therapeutic drugs, agricultural chemicals, food additives and contaminants, metals, solvents, combustion products, cosmetics, and toxins. Some of these, such as combustion products, are the products of use processes rather than being use classes. All of these groups of chemicals are discussed in detail in Chapter 4.

Movement of toxicants in the environment

Chemicals released into the environment rarely remain in the form, or at the location, of release. For example, agricultural chemicals used as sprays may drift from the point of application as air contaminants or enter run-off water as water contaminants. Many of these chemicals are susceptible to fungal or bacterial degradation and are rapidly detoxified, frequently being broken down to products that can enter the carbon, nitrogen, and oxygen cycles. Other agricultural chemicals, particularly halogenated organic compounds, are recalcitrant to a greater or lesser degree to metabolism by microorganisms and persist in soil and water as contaminants; they may enter biologic food chains and move to higher trophic levels or persist in processed crops as food contaminants. This same scenario is applicable to any toxicant released into the environment either for a specific use or as a result of industrial processes,

combustion, and so on. Chemicals released into the environment are also susceptible to chemical degradation, a process often stimulated by ultraviolet light.

Although most transport between inanimate phases of the environment results in wider dissemination, but, at the same time, dilution of the toxicant in question, transfer between living creatures may result in increased concentration or bioaccu-mulation. Lipid-soluble toxicants are readily taken up by organisms following exposure in air, water, or soil. Unless rapidly metabolized, they persist in the tissues long enough to be transferred to the next trophic level. At each level, the lipophilic toxicant tends to be retained while the bulk of the food is digested, utilized, and excreted, thus increasing the toxicant concentration. At some point in the chain, the toxicant can become deleterious, particularly if the organism at that level is more susceptible than those at the level preceding it. Thus, the eggshell thinning in certain raptorial birds was almost certainly due to the uptake of DDT (1,1,1-trichloro-2,2-bis(4-chlorophenyl) ethane) and DDE (1,1-dichloro-2,2-bis(4-chlorophenyl) ethane) and their particular susceptibility to this type of toxicity.

It is clear that such transport can occur through both aquatic and terrestrial food chains, although in the former, higher members of the chains, such as fish, can accumulate large amounts of toxicants directly from the medium. This accumulation occurs because of the large area of gill filaments, their intimate contact with the water, and the high flow rate of water over them. Given these characteristics and a toxicant with a high partition coefficient between lipid membranes and water, considerable uptake is inevitable.

CHAPTER 2
NUCLEAR EXPLOSION

Nuclear explosions produce both immediate and delayed destructive effects. Immediate effects (blast, thermal radiation, prompt ionizing radiation) are produced and cause significant destruction within seconds or minutes of a nuclear detonation. The delayed effects (radioactive fallout and other possible environmental effects) inflict damage over an extended period ranging from hours to centuries, and can cause adverse effects in locations very distant from the site of the detonation. These two classes of effects are treated in separate subsections.

Overview of Immediate Effects

The three categories of immediate effects are: blast, thermal radiation (heat), and prompt ionizing or nuclear radiation. Their relative importance varies with the yield of the bomb. At low yields, all three can be significant sources of injury. With an explosive yield of about 2.5 kt, the three effects are roughly equal. All are capable of inflicting fatal injuries at a range of 1 km.

The equations below provide approximate scaling laws for relating the destructive radius of each effect with yield:

```
r_thermal = Y^0.41 * constant_th
r_blast = Y^0.33 * constant_bl
r_radiation = Y^0.19 * constant_rad
```

If Y is in multiples (or fractions) of 2.5 kt, then the result is in km (and all the constants equal one). This is based on thermal radiation just sufficient to cause 3rd degree burns (8 calories/cm^2); a 4.6 psi blast overpressure (and optimum burst height); and a 500 rem radiation dose.

The underlying principles behind these scaling laws are easy to explain. The fraction of a bomb's yield emitted as thermal radiation, blast, and ionizing radiation are essentially constant for all yields, but the way the different forms of energy interact with air and targets vary dramatically.

Air is essentially transparent to thermal radiation. The thermal radiation affects exposed surfaces, producing damage by rapid heating. A bomb that is 100 times larger can produce equal thermal radiation intensities over areas 100 times larger. The area of an (imaginary) sphere centered on the explosion increases with the square of the radius. Thus the destructive radius increases with the square root of the yield (this is the familiar inverse square law of electromagnetic radiation). Actually the rate of increase is somewhat less, partly due to the fact that larger bombs emit heat more slowly which reduces the damage produced by each calorie of heat. It is important to note that the area subjected to damage by thermal radiation increases almost linearly with yield.

Blast effect is a volume effect. The blast wave deposits energy in the material it passes through, including air. When the blast wave passes through solid material, the energy left behind causes damage. When it passes through air it simply grows weaker. The more matter the energy travels through, the smaller the effect. The amount of matter increases with the volume of the imaginary sphere centered on the explosion. Blast effects thus scale with the inverse cube law which relates radius to volume.

The intensity of nuclear radiation decreases with the inverse square law like thermal radiation. However nuclear radiation is also strongly absorbed by the air it travels through, which causes the intensity to drop off much more rapidly.

These scaling laws show that the effects of thermal radiation grow rapidly with yield (relative to blast), while those of radiation rapidly decline.

In the Hiroshima attack (bomb yield approx. 15 kt) casualties (including fatalities) were seen from all three causes. Burns (including those caused by the ensuing fire storm) were the most prevalent serious injury (two thirds of those who died the first day were burned), and occurred at the greatest range. Blast and burn injuries were both found in 60–70 % of all survivors. People close enough to suffer significant radiation illness were well inside the lethal effects radius for blast and flash burns, as a result only 30 % of injured survivors showed radiation illness. Many of these people were sheltered from burns and

blast and thus escaped their main effects. Even so, most victims with radiation illness also had blast injuries or burns as well.

With yields in the range of hundreds of kilotons or greater (typical for strategic warheads) immediate radiation injury becomes insignificant. Dangerous radiation levels only exist so close to the explosion that surviving the blast is impossible. On the other hand, fatal burns can be inflicted well beyond the range of substantial blast damage. A 20 megaton bomb can cause potentially fatal third degree burns at a range of 40 km, where the blast can do little more than break windows and cause superficial cuts.

It should be noted that the atomic bombings of Hiroshima and Nagasaki caused fatality rates were one to two orders of magnitude higher than the rates in conventional fire raids on other Japanese cities. Eventually on the order of 200,000 fatalities, which is about one-quarter of all Japanese bombing deaths, occurred in these two cities with a combined population of less than 500,000. This is due to the fact that the bombs inflicted damage on people and buildings virtually instantaneously and without warning, and did so with the combined effects of flash, blast, and radiation. Widespread fatal injuries were thus inflicted instantly, and the many more people were incapacitated and thus unable to escape the rapidly developing fires in the suddenly ruined cities. Fire raids in comparison, inflicted few immediate or direct casualties; and a couple of hours elapsed from the raid's beginning to the time when conflagrations became general, during which time the population could flee.

A convenient rule of thumb for estimating the short-term fatalities from all causes due to a nuclear attack is to count everyone inside the 5 psi blast overpressure contour around the hypocenter as a fatality. In reality, substantial numbers of people inside the contour will survive and substantial numbers outside the contour will die, but the assumption is that these two groups will be roughly equal in size and balance out. This completely ignores any possible fallout effects.

Overview of Delayed Effects

Radioactive Contamination

The chief delayed effect is the creation of huge amounts of radioactive material with long lifetimes (half-lifes ranging from days to millennia). The primary source of these products is the debris left from fission reactions. A potentially significant secondary source is neutron capture by non-radioactive isotopes both within the bomb and in the outside environment.

When atoms fission they can split in some 40 different ways, producing a mix of about 80 different isotopes. These isotopes vary widely in stability, some our completely stable while others undergo radioactive decay with half-lifes of fractions of a second. The decaying isotopes may themselves form stable or unstable daughter isotopes. The mixture thus quickly becomes even more complex, some 300 different isotopes of 36 elements have been identified in fission products.

Short-lived isotopes release their decay energy rapidly, creating intense radiation fields that also decline quickly. Long-lived isotopes release energy over long periods of time, creating radiation that is much less intense but more persistent. Fission products thus initially have a very high level of radiation that declines quickly, but as the intensity of radiation drops, so does the rate of decline.

A useful rule-of-thumb is the "rule of sevens". This rule states that for every seven-fold increase in time following a fission detonation (starting at or after 1 hour), the radiation intensity decreases by a factor of 10. Thus after 7 hours, the residual fission radioactivity declines 90 %, to one-tenth its level of 1 hour. After 7*7 hours (49 hours, approx. 2 days), the level drops again by 90 %. After 7*2 days (2 weeks) it drops a further 90 %; and so on for 14 weeks. The rule is accurate to 25 % for the first two weeks, and is accurate to a factor of two for the first six months. After 6 months, the rate of decline becomes much more rapid. The rule of sevens corresponds to an approximate t^-1.2 scaling relationship.

These radioactive products are most hazardous when they settle to the ground as «fallout». The rate at which fallout settles depends very strongly on the altitude at which the explosion occurs, and to a lesser extent on the size of the explosion.

If the explosion is a true air-burst (the fireball does not touch the ground), when the vaporized radioactive products cool enough to condense and solidify, they will do so to form microscopic particles. These particles are mostly lifted high into the atmosphere by the rising fireball, although significant amounts are deposited in the lower atmosphere by mixing that occurs due to convective circulation within the fireball. The larger the explosion, the higher and faster the fallout is lofted, and the smaller the proportion that is deposited in the lower atmosphere. For explosions with yields of 100 kt or less, the fireball does not rise abve the troposphere where precipitation occurs. All of this fallout will thus be brought to the ground by weather processes within months at most (usually much faster). In the megaton range, the fireball rises so high that it enters the stratosphere. The stratosphere is dry, and no weather processes exist there to bring fallout down quickly. Small fallout particles will descend over a period of months or years. Such long-delayed fallout has lost most of its hazard by the time it comes down, and will be distributed on a global scale. As yields increase above 100 kt, progressively more and more of the total fallout is injected into the stratosphere.

An explosion closer to the ground (close enough for the fireball to touch) sucks large amounts of dirt into the fireball. The dirt usually does not vaporize, and if it does, there is so much of it that it forms large particles. The radioactive isotopes are deposited on soil particles, which can fall quickly to earth. Fallout is deposited over a time span of minutes to days, creating downwind contamination both nearby and thousands of kilometers away. The most intense radiation is created by nearby fallout, because it is more densely deposited, and because short-lived isotopes haven't decayed yet. Weather conditions can affect this con-

siderably of course. In particular, rainfall can «rain out» fallout to create very intense localized concentrations. Both external exposure to penetrating radiation, and internal exposure (ingestion of radioactive material) pose serious health risks.

Explosions close to the ground that do not touch it can still generate substantial hazards immediately below the burst point by neutron-activation. Neutrons absorbed by the soil can generate considerable radiation for several hours.

The megaton class weapons that were developed in the US and USSR during the fifties and sixties have been largely retired, being replaced with much smaller yield warheads. The yield of a modern strategic warhead is, with few exceptions, now typically in the range of 200–750 kt. Recent work with sophisticated climate models has shown that this reduction in yield results in a much larger proportion of the fallout being deposited in the lower atmosphere, and a much faster and more intense deposition of fallout than had been assumed in studies made during the sixties and seventies. The reduction in aggregate strategic arsenal yield that occurred when high yield weapons were retired in favor of more numerous lower yield weapons has actually increased the fallout risk.

Effects on the Atmosphere and Climate

Although not as directly deadly as fallout, other environmental effects can be quite harmful.

Harm to the Ozone Layer

The high temperatures of the nuclear fireball, followed by rapid expansion and cooling, cause large amounts of nitrogen oxides to form from the oxygen and nitrogen in the atmosphere (very similar to what happens in combustion engines). Each megaton of yield will produce some 5000 tons of nitrogen oxides. The rising fireball of a high kiloton or megaton range warhead will carry these nitric oxides well up into the stratosphere, where they can reach the ozone layer. A series of large atmospheric explosions could significantly deplete the ozone layer. The high yield tests in the fifties and sixties probably did cause significant depletion, but the ozone measurements made at the time were too limited to pick up the expected changes out of natural variations.

Nuclear Winter

The famous TTAPS (Turco, Toon, Ackerman, Pollack, and Sagan) proposal regarding a potential «nuclear winter» is another possible occurrence. This effect is caused by the absorption of sunlight when large amounts of soot are injected into the atmosphere by the widespread burning of cities and petroleum stocks destroyed in a nuclear attack.

Similar events have been observed naturally when large volcanic eruptions

have injected large amounts of dust into the atmosphere. The Tambora eruption of 1815 (the largest volcanic eruption in recent history) was followed by «the year without summer» in 1816, the coldest year in the last few centuries.

Soot is far more efficient in absorbing light than volcanic dust, and soot particles are small and hydrophobic and thus tend not to settle or wash out as easily.

Although the initial TTAPS study was met with significant skepticism and criticism, later and more sophisticated work by researchers around the world have confirmed it in all essential details. These studies predict that the amount of soot that would be produced by burning most of the major cities in the US and USSR would severly disrupt climate on a world-wide basis. The major effect would be a rapid and drastic reduction in global temperature, especially over land. All recent studies indicate that if large scale nucelar attack occur against urban or petrochemical targets, average temperature reductions of at least 10 degrees C would occur lasting many months. This level of cooling far exceeds any that has been observed in recorded history, and is comparable to that of a full scale ice age. In areas downwind from attack sites, the cooling can reach 35 degrees C. It is probable that no large scale temperature excursion of this size has occurred in 65 million years.

Smaller attacks would create reduced effects of course. But it has been pointed out that most of the world's food crops are subtropical plants that would have dramatic drops in productivity if an average temperature drop of even one degree were to occur for even a short time during the growing season. Since the world maintains a stored food supply equal to only a few months of consumption, a war during the Northern Hemisphere spring or summer could still cause deadly starvation around the globe from this effect alone even if it only produced a mild «nuclear autumn».

Physics of Nuclear Weapon Effects

Thermal radiation and blast are inevitable consequences of the near instantaneous release of an immense amount of energy in a very small volume, and are thus characteristic to all nuclear weapons regardless of type or design details. The release of ionizing radiation, both at the instant of explosion and delayed radiation from fallout, is governed by the physics of the nuclear reactions involved and how the weapon is constructed, and is thus very dependent on both weapon type and design.

Fireball Physics

The fireball is the hot ball of gas created when a nuclear explosion heats the bomb itself, and the immediate surrounding environment, to very high temperatures. As this incandescent ball of hot gas expands, it radiates part of its energy away as thermal radiation (including visible and ultraviolet light), part of its energy also goes into creating a shock wave or blast wave in the surrounding

environment. The generation of these two destructive effects are thus closely linked by the physics of the fireball. In the discussion below I assume the fireball is forming in open air, unless stated otherwise.

The Early Fireball

Immediately after the energy-producing nuclear reactions in the weapon are completed, the energy is concentrated in the nuclear fuels themselves. The energy is stored as (in order of importance): thermal radiation or photons; as kinetic energy of the ionized atoms and the electrons (mostly as electron kinetic energy since free electrons outnumber the atoms); and as excited atoms, which are partially or completely stripped of electrons (partially for heavy elements, completely for light ones).

Thermal (also called blackbody) radiation is emitted by all matter. The intensity and most prevalent wavelength is a function of the temperature, both increasing as temperature increases. The intensity of thermal radiation increases very rapidly — as the fourth power of the temperature. Thus at the 60–100 million degrees C of a nuclear explosion, which is some 10,000 times hotter than the surface of the sun, the brightness (per unit area) is some 10 quadrillion (10^16) times greater! Consequently about 80 % of the energy in a nuclear explosion exists as photons. At these temperatures the photons are soft x-rays with energies in the range of 10–200 KeV.

The first energy to escape from the bomb are the gamma rays produced by the nuclear reactions. They have energies in the MeV range, and a significant number of them penetrate through the tampers and bomb casing and escape into the outside world at the speed of light. The gamma rays strike and ionize the surrounding air molecules, causing chemical reactions that form a dense layer of «smog» tens of meters deep around the bomb. This smog is composed primarily of ozone, and nitric and nitrous oxides.

X-rays, particularly the ones at the upper end of the energy range, have substantial penetrating power and can travel significant distances through matter at the speed of light before being absorbed. Atoms become excited when they absorb x-rays, and after a time they re-emit part of the energy as a new lower energy x-ray. By a chain of emissions and absorptions, the x-rays carry energy out of the hot center of the bomb, a process called radiative transport. Since each absorption/re-emission event takes a certain amount of time, and the direction of re-emission is random (as likely back toward the center of the bomb as away from it), the net rate of radiative transport is considerably slower than the speed of light. It is however initially much faster than the expansion of the plasma (ionized gas) making up the fireball or the velocity of the neutrons.

An expanding bubble of very high temperatures is thus formed called the «iso-thermal sphere». It is a sphere were everything has been heated by x-rays to a nearly uniform temperature, initially in the tens of millions of degrees. As soon as the sphere expands beyond the bomb casing it begins radiating light

away through the air (unless the bomb is buried or underwater). Due to the still enormous temperatures, it is incredibly brilliant (surface brightness trillions of times more intense than the sun). Most of the energy being radiated is in the x-ray and far ultraviolet range to which air is not transparent. Even at the wavelengths of the near ultraviolet and visible light, the «smog» layer absorbs much of the energy. Then too, at this stage the fireball is only a few meters across. Thus the apparent surface brightness at a distance, and the output power (total brightness) is not nearly as intense as the fourth-power law would indicate.

Blast Wave Development and Thermal Radiation Emission

As the fireball expands, it cools and the wavelength of the photons transporting energy drops. Longer wavelength photons do not penetrate as far before being absorbed, so the speed of energy transport also drops. When the isothermal sphere cools to about 300,000 degrees C (and the surface brightness has dropped to being a mere 10 million times brighter than the sun), the rate of radiative growth is about equal to the speed of sound in the fireball plasma. At this point a shock wave forms at the surface of the fireball as the kinetic energy of the fast moving ions starts transferring energy to the surrounding air. This phenomenon, known as «hydrodynamic separation», occurs for a 20 kt explosion about 100 microseconds after the explosion, when the fireball is some 13 meters across. A shock wave internal to the fireball caused by the rapidly expanding bomb debris may overtake and reinforce the fireball surface shock wave a few hundred microseconds later.

The shock wave initially moves at some 30 km/sec, a hundred times the speed of sound in normal air. This compresses and heats the air enormously, up to 30,000 degrees C (some five times the sun's surface temperature). At this temperature the air becomes ionized and incandescent. Ionized gas is opaque to visible radiation, so the glowing shell created by the shock front hides the much hotter isothermal sphere inside. The shock front is many times brighter than the sun, but since it is much dimmer than the isothermal sphere it acts as an optical shutter, causing the fireball's thermal power to drop rapidly.

The fireball is at its most brilliant just as hydrodynamic separation occurs, the great intensity compensating for the small size of the fireball. The rapid drop in temperature causes the thermal power to drop ten-fold, reaching a minimum in about 10 milliseconds for a 20 kt bomb (100 milliseconds for 1 Mt bomb). This «first pulse» contains only about 1 percent of the bomb's total emitted thermal radiation. At this minimum, the fireball of a 20 kt bomb is 180 meters across.

As the shock wave expands and cools to around 3000 degrees, it stops glowing and gradually also becomes transparent. This is called «breakaway» and occurs at about 15 milliseconds for a 20 kt bomb, when the shock front has expanded to 220 meters and is travelling at 4 km/second. The isothermal sphere, at a still very luminous 8000 degrees, now becomes visible and both the apparent sur-

face temperature and brightness of the fireball climb to form the «second pulse». The isothermal sphere has grown considerably in size and now consists almost entirely of light at wavelengths to which air is transparent, so it regains much of the total luminosity of the first peak despite its lower temperature. This second peak occurs at 150 milliseconds for a 20 kt bomb, at 900 milliseconds for a 1 Mt bomb. After breakaway, the shock (blast) wave and the fireball do not interact further.

A firm cutoff for this second pulse is impossible to provide because the emission rate gradually declines over an extended period. Some rough guidelines are that by 300 milliseconds for a 20 kt bomb (1.8 seconds for a 1 Mt) 50 % of the total thermal radiation has been emitted, and the rate has dropped to 40 % of the second peak. These figures become 75 % total emitted and 10 % peak rate by 750 milliseconds (20 kt) and 4.5 second (1 Mt). The emission time scales roughly as the 0.45 power of yield (Y^0.45).

Although this pulse never gets as bright as the first, it emits about 99 % of the thermal radiation because it is so much longer.

Ionizing Radiation Physics

There are four types of ionizing radiation produced by nuclear explosions that can cause significant injury: neutrons, gamma rays, beta particles, and alpha particles. Gamma rays are energetic (short wavelength) photons (as are X-rays), beta particles are energetic (fast moving) electrons, and alpha particles are energetic helium nuclei. Neutrons are damaging whether they are energetic or not, although the faster they are, the worse their effects.

They all share the same basic mechanism for causing injury though: the creation of chemically reactive compounds called «free radicals» that disrupt the normal chemistry of living cells. These radicals are produced when the energetic radiation strikes a molecule in the living issue, and breaks it into ionized (electrically charged) fragments. Fast neutrons can do this also, but all neutrons can also transmute ordinary atoms into radioactive isotopes, creating even more ionizing radiation in the body.

The different types of radiation present different risks however. Neutrons and gamma rays are very penetrating types of radiation. They are the hardest to stop with shielding. They can travel through hundreds of meters of air and the walls of ordinary houses. They can thus deliver deadly radiation doses even if an organism is not in immediate contact with the source. Beta particles are less penetrating, they can travel through several meters of air, but not walls, and can cause serious injury to organisms that are near to the source. Alpha particles have a range of only a few centimeters in air, and cannot even penetrate skin. Alphas can only cause injury if the emitting isotope is ingested.

The shielding effect of various materials to radiation is usually expressed in half-value thickness, or tenth-value thickness: in other words, the thickness of material required to reduce the intensity of radiation by one-half or one-tenth.

Successive layers of shielding each reduce the intensity by the same proportion, so three tenth-value thickness reduce the intensity to one-thousandth (a tenth-value thickness is about 3.3 half-value thicknesses). Some example tenth-value thicknesses for gamma rays are: steel 8.4–11 cm, concrete 28–41 cm, earth 41–61 cm, water 61–100 cm, and wood 100–160 cm. The thickness ranges indicate the varying shielding effect for different gamma ray energies.

Even light clothing provides substantial shielding to beta rays.

Sources of Radiation

Prompt Radiation

Radiation is produced directly by the nuclear reactions that generate the explosion, and by the decay of radioactive products left over (either fission debris, or induced radioactivity from captured neutrons).

The explosion itself emits a very brief burst (about 100 nanoseconds) of gamma rays and neutrons, before the bomb has blown itself apart. The intensity of these emissions depends very heavily on the type of weapon and the specific design. In most designs the initial gamma ray burst is almost entirely absorbed by the bomb (tamper, casing, explosives, etc.) so it contributes little to the radiation hazard. The neutrons, being more penetrating, may escape. Both fission and fusion reactions produce neutrons. Fusion produces many more of them per kiloton of yield, and they are generally more energetic than fission neutrons. Some weapons (neutron bombs) are designed specifically to emit as much energy in the form as neutrons as possible. In heavily tamped fission bombs few if any neutrons escape. It is estimated that no significant neutron exposure occurred from Fat Man, and only 2 % of the total radiation dose from Little Boy was due to neutrons.

The neutron burst itself can be a significant source of radiation, depending on weapon design. As the neutrons travel through the air they are slowed by collisions with air atoms, and are eventually captured. Even this process of neutron attenuation generates hazardous radiation. Part of the kinetic energy lost by fast neutrons as they slow is converted into gamma rays, some with very high energies (for the 14.1 MeV fusion neutrons). The duration of production for these neutron scattering gammas is about 10 microseconds. The capture of neutrons by nitrogen-14 also produces gammas, a process completed by 100 milliseconds.

Immediately after the explosion, there are substantial amounts of fission products with very short half-lifes (milliseconds to minutes). The decay of these isotopes generate correspondingly intense gamma radiation that is emitted directly from the fireball. This process is essentially complete within 10 seconds.

The relative importance of these gamma ray sources depends on the size of the explosion. Small explosions (20 kt, say) can generate up to 25 % of the gamma dose from the direct gammas and neutron reactions. For large explosions (1 Mt) this contribution is essentially zero. In all cases, the bulk of the gammas

are produced by the rapid decay of radioactive debris.

Delayed Radiation

Radioactive decay is the sole source of beta and alpha particles. They are also emitted during the immediate decay mentioned above of course, but their range is too short to make any prompt radiation contribution. Betas and alphas become important when fallout begins settling out. Gammas remain very important at this stage as well.

Fallout is a complex mixture of different radioactive isotopes, the composition of which continually changes as each isotope decays into other isotopes. Many isotopes make significant contributions to the overall radiation level. Radiation from short lived isotopes dominates initially, and the general trend is for the intensity to continually decline as they disappear. Over time the longer lived isotopes become increasingly important, and a small number of isotopes emerge as particular long-term hazards.

Radioactive isotopes are usually measured in terms of curies. A curie is the quantity of radioactive material that undergoes 3.7×10^{10} decays/sec (equal to 1 g of radium-226). More recently the SI unit bequerel has become common in scientific literature, one bequerel is 1 decay/sec. The fission of 57 grams of material produces 3×10^23 atoms of fission products (two for each atom of fissionable material). One minute after the explosion this mass is undergoing decays at a rate of 10^21 disintegrations/sec (3x10^10 curies). It is estimated that if these products were spread over 1 km^2, then at a height of 1 m above the ground one hour after the explosion the radiation intensity would be 7500 rads/hr.

Isotopes of special importance include iodine-131, strontium-90 and 89, and cesium-137. This is due to both their relative abundance in fallout, and to their special biological affinity. Isotopes that are readily absorbed by the body, and concentrated and stored in particular tissues can cause harm out of proportion to their abundance.

Iodine-131 is a beta and gamma emitter with a half-life of 8.07 days (specific activity 124,000 curies/g) Its decay energy is 970 KeV; usually divided between 606 KeV beta, 364 KeV gamma. Due to its short half-life it is most dangerous in the weeks immediately after the explosion, but hazardous amounts can persist for a few months. It constitutes some 2 % of fission-produced isotopes — 1.6×10^5 curies/kt. Iodine is readily absorbed by the body and concentrated in one small gland, the thyroid.

Strontium-90 is a beta emitter (546 KeV, no gammas) with a half-life of 28.1 years (specific activity 141 curies/g), Sr-89 is a beta emitter (1.463 MeV, gammas very rarely) with a half-life of 52 days (specific activity 28,200 Ci/g). Each of these isotopes constitutes about 3 % of total fission isotopes: 190 curies of Sr-90 and 3.8×10^4 curies of Sr-89 per kiloton. Due to their chemical resemblance to calcium these isotopes are absorbed fairly well, and stored in bones. Sr-89 is an important hazard for a year or two after an explosion, but Sr-90 re-

mains a hazard for centuries. Actually most of the injury from Sr-90 is due to its daughter isotope yttrium-90. Y-90 has a half-life of only 64.2 hours, so it decays as fast as it is formed, and emits 2.27 MeV beta particles.

Cesium-137 is a beta and gamma emitter with a half-life of 30.0 years (specific activity 87 Ci/g). Its decay energy is 1.176 MeV; usually divided by 514 KeV beta, 662 KeV gamma. It comprises some 3–3.5 % of total fission products — 200 curies/kt. It is the primary long-term gamma emitter hazard from fallout, and remains a hazard for centuries.

Although not important for acute radiation effects, the isotopes carbon-14 and tritium are also of interest because of possible genetic injury. These are not direct fission products. They are produced by the interaction of fission and fusion neutrons with the atmosphere and, in the case of tritium, as a direct product of fusion reactions. Most of the tritium generated by fusion is consumed in the explosion but significant amounts survive. Tritium is also formed by the capture of fast neutrons by nitrogen atoms in the air: N-14 + n -> T + C-12. Carbon-14 in also formed by neutron-nitrogen reactions: N-14 + n -> C-14 + p. Tritium is a very weak beta emitter (18.6 KeV, no gamma) with a half-life of 12.3 years (9700 Ci/g).

Carbon-14 is also a weak beta emitter (156 KeV, no gamma), with a half-life of 5730 years (4.46 Ci/g). Atmospheric testing during the fifties and early sixties produced about 3.4 g of C-14 per kiloton (15.2 curies) for a total release of 1.75 tonnes (7.75×10^6 curies). For comparison, only about 1.2 tonnes of C-14 naturally exists, divided between the atmosphere (1 tonne) and living matter (0.2 tonne). Another 50-80 tonnes is dissolved in the oceans. Due to carbon exchange between the atmosphere and oceans, the half-life of C-14 residing in the atmosphere is only about 6 years. By now the atmospheric concentration has returned to within 1 % or so of normal. High levels of C-14 remain in organic material formed during the sixties (in wood, say, or DNA).

Electromagnetic Effects

The high temperatures and energetic radiation produced by nuclear explosions also produce large amounts of ionized (electrically charged) matter which is present immediately after the explosion. Under the right conditions, intense currents and electromagnetic fields can be produced, generically called EMP (Electromagnetic Pulse), that are felt at long distances. Living organisms are impervious to these effects, but electrical and electronic equipment can be temporarily or permanently disabled by them. Ionized gases can also block short wavelength radio and radar signals (fireball blackout) for extended periods.

The occurrence of EMP is strongly dependent on the altitude of burst. It can be significant for surface or low altitude bursts (below 4,000 m); it is very significant for high altitude bursts (above 30,000 m); but it is not significant for altitudes between these extremes. This is because EMP is generated by the asymmetric absorption of instantaneous gamma rays produced by the explosion. At intermediate altitudes the air absorbs these rays fairly uniformly and does not

generate long range electromagnetic disturbances.

Mechanisms of Damage and Injury

The different mechanisms are discussed individually, but it should be no surprise that in combination they often accentuate the harm caused by each other. I will discuss such combined effects wherever appropriate.

Thermal Damage and Incendiary Effects

Thermal damage from nuclear explosions arises from the intense thermal (heat) radiation produced by the fireball. The thermal radiation (visible and infrared light) falls on exposed surfaces and is wholly or partly absorbed. The radiation lasts from about a tenth of a second, to several seconds depending on bomb yield (it is longer for larger bombs). During that time its intensity can exceed 1000 watts/cm^2 (the maximum intensity of direct sunlight is 0.14 watts/cm^2). For a rough comparison, the effect produced is similar to direct exposure to the flame of an acetylene torch.

The heat is absorbed by the opaque surface layer of the material on which it falls, which is usually a fraction of a millimeter thick. Naturally dark materials absorb more heat than light colored or reflective ones. The heat is absorbed much faster than it can be carried down into the material through conduction, or removed by reradiation or convection, so very high temperatures are produced in this layer almost instantly. Surface temperatures can exceed 1000 degrees C close to the fireball. Such temperatures can cause dramatic changes to the material affected, but they do not penetrate in very far.

More total energy is required to inflict a given level of damage for a larger bomb than a smaller one since the heat is emitted over a longer period of time, but this is more than compensated for by the increased thermal output. The thermal damage for a larger bomb also penetrates further due to the longer exposure.

Thermal radiation damage depends very strongly on weather conditions. Cloud cover, smoke, or other obscuring material in the air can considerably reduce effective damage ranges over clear air conditions.

For all practical purposes, the emission of thermal radiation by a bomb is complete by the time the shock wave arrives. Regardless of yield, this generalization is only violated in the area of total destruction around a nuclear explosion where 100 % mortality would result from any one of the three damage effects.

Incendiary effects refer to anything that contributes to the occurrence of fires after the explosion, which is a combination of the effects of thermal radiation and blast.

First degree flash burns are not serious, no tissue destruction occurs. They are characterized by immediate pain, followed by reddening of the skin. Pain and sensitivity continues for some minutes or hours, after which the affected skin returns to normal without further incident.

Second degree burns cause damage to the underlying dermal tissue, killing

some portion of it. Pain and redness is followed by blistering within a few hours as fluids collect between the epidermis and damaged tissue. Sufficient tissue remains intact however to regenerate and heal the burned area quickly, usually without scarring. Broken blisters provide possible infection sites prior to healing.

Third degree burns cause tissue death all the way through the skin, including the stem cells required to regenerate skin tissue. The only way a 3rd degree burn can heal is by skin regrowth from the edges, a slow process that usually results in scarring, unless skin grafts are used. Before healing 3rd degree burns present serious risk of infection, and can cause serious fluid loss. A 3rd degree burn over 25 % of the body (or more) will typically precipitate shock in minutes, which itself requires prompt medical attention.

Even more serious burns are possible, which have been classified as fourth (even fifth) degree burns. These burns destroy tissue below the skin: muscle, connective tissue etc. They can be caused by thermal radiation exposures substantially in excess of those in the table for 3rd degree burns. Many people close to the hypocenter of the Hiroshima bomb suffered these types of burns. In the immediate vicinity of ground zero the thermal radiation exposure was 100 c/cm^2, some fifteen times the exposure required for 3rd degree burns, most of it within the first 0.3 seconds (which was the arrival time of the blast wave). This is sufficient to cause exposed flesh to flash into steam, flaying exposed body areas to the bone.

At the limit of the range for 3rd degree burns, the time lapse between suffering burns and being hit by the blast wave varies from a few seconds for low kiloton explosions to a minute of so for high megaton yields.

Incendiary Effects

Despite the extreme intensity of thermal radiation, and the extraordinary surface temperatures that occur, it has less incendiary effect than might be supposed. This is mostly due to its short duration, and the shallow penetration of heat into affected materials. The extreme heating can cause pyrolysis (the charring of organic material, with the release of combustible gases), and momentary ignition, but it is rarely sufficient to cause self-sustained combustion. This occurs only with tinder-like, or dark, easily flammable materials: dry leaves, grass, old newspaper, thin dark flammable fabrics, tar paper, etc. The incendiary effect of the thermal pulse is also substantially affected by the later arrival of the blast wave, which usually blows out any flames that have already been kindled. Smoldering material can cause reignition later however.

The major incendiary effect of nuclear explosions is caused by the blast wave. Collapsed structures are much more vulnerable to fire than intact ones. The blast reduces many structures to piles of kindling, the many gaps opened in roofs and walls act as chimneys, gas lines are broken open, storage tanks for flammable materials are ruptured. The primary ignition sources appear to be flames and pilot lights in heating appliances (furnaces, water heaters, stoves, ov-

ens, etc.). Smoldering material from the thermal pulse can be very effective at igniting leaking gas.

Although the ignition sources are probably widely scattered a number of factors promote their spread into mass fires. The complete suppression of fire fighting efforts is extremely important. Another is that the blast scatters combustible material across fire breaks that normally exist (streets, yards, fire lanes, etc.).

The effectiveness of building collapse, accompanied by the disruption of fire fighting, in creating mass fires can be seen in the San Francisco earthquake (1906), the Tokyo-Yokahama earthquake (1923), and the recent Kobe earthquake (1995). In these disasters there was no thermal radiation to ignite fires, and the scattering of combustible materials did not occur, but huge fires still resulted. In San Francisco and Tokyo-Yokohama these fires were responsible for most of the destruction that occurred.

In Hiroshima the fires developed into a true firestorm. This is an extremely intense fire that produces a rapidly rising column of hot air over the fire area, in turn powerful winds are generated which blow in to the fire area, fanning and feeding the flames. The fires continue until all combustible material is exhausted. Firestorms develop from multiple ignition sources spread over a wide area that create fires which coalesce into one large fire. Temperatures in firestorm areas can reach many hundreds of degrees, carbon monoxide reaches lethal levels, few people who see the interior of a firestorm live to tell about it. Firestorms can melt roads, cars, and glass. They can boil water in lakes and rivers, and cook people to death in buried bomb shelters. The in-blowing winds can reach gale force, but they also prevent the spread of the fires outside of the area in which the firestorm initially develops. The firestorm in Hiroshima began only about 20 minutes after the bombing.

Nagasaki did not have a firestorm, instead it had a type of mass fire called a conflagration. This is a less intense type of fire, it develops and burns more slowly. A conflagration can begin in multiple locations, or only one. Conflagrations can spread considerable distances from their origins. The fires at Nagasaki took about 2 hours to become well established, and lasted 4–5 hours.

Eye Injury

The brightness and thermal output of a nuclear explosion presents an obvious source of injury to the eye. Injury to the cornea through surface heating, and injury to the retina are both possible risks. Surprisingly, very few cases of injury were noted in Japan. A number of factors acted to reduce the risk. First, eye injury occurs when vision is directed towards the fireball. People spend relatively little time looking up at the sky so only a very small portion of the population would have their eyes directed at the fireball at the time of burst. Second, since the bomb exploded in bright daylight the eye pupil would be expected to be small.

About 4 % of the population within the 3rd degree burn zone at Hiroshima reported keratitis, pain and inflammation of the cornea, which lasted several hours to several days. No other corneal damage was noted.

The most common eye injury was flashblindness, a temporary condition in which the visual pigment of retina is bleached out by the intense light. Vision is completely recovered as the pigment is regenerated, a process that takes several seconds to several minutes. This can cause serious problems though in carrying out emergency actions, like taking cover from the oncoming blast wave.

Retinal injury is the most far reaching injury effect of nuclear explosions, but it is relatively rare since the eye must be looking directly at the detonation. Retinal injury results from burns in the area of the retina where the fireball image is focused. The brightness per unit area of a fireball does not diminish with distance (except for the effects of haze), the apparent fireball size simply gets smaller. Retinal injury can thus occur at any distance at which the fireball is visible, though the affected area of the retina gets smaller as range increases. The risk of injury is greater at night since the pupil is dialated and admits more light. For explosions in the atmosphere of 100 kt and up, the blink reflex protects the retina from much of the light.

Blast Damage and Injury

Blast damage is caused by the arrival of the shock wave created by the nuclear explosion. Shock waves travel faster than sound, and cause a virtually instantaneous jump in pressure at the shock front. The air immediately behind the shock front is accelerated to high velocities and creates a powerful wind. The wind in turn, creates dynamic pressure against the side of objects facing the blast. The combination of the pressure jump (called the overpressure)and the dynamic pressure causes blast damage.

Both the overpressure and dynamic pressure jump immediately to their peak values when the shock wave arrives. They then decay over a period ranging from a few tenths of a second to several seconds, depending on the strength of the blast and the yield. Following the this there is a longer period of weaker negative pressure before the atmospheric conditions return to normal. The negative pressure has little significance as far as causing damage or injury is concerned. A given pressure is more destructive from a larger bomb, due its longer duration.

The is a definite relationship between the overpressure and the dynamic pressure. The overpressure and dynamic pressure are equal at 70 psi, and the wind speed is 1.5 times the speed of sound. Below an overpressure of 70 psi, the dynamic pressure is less than the overpressure; above 70 psi it exceeds the overpressure. Since the relationship is fixed it is convenient to use the overpressure alone as a yardstick for measuring blast effects. At 20 psi overpressure the wind speed is still 500 mph, higher than any tornado wind.

As a general guide, city areas are completely destroyed (with massive loss

of life) by overpressures of 5 psi, with heavy damage extending out at least to the 3 psi contour. The dynamic pressure is much less than the overpressure at blast intensities relevant for urban damage, although at 5 psi the wind speed is still 162 mph — close to the peak wind speeds of the most intense hurricanes.

Humans are actually quite resistant to the direct effect of overpressure. Pressures of over 40 psi are required before lethal effects are noted. This pressure resistance makes it possible for unprotected submarine crews to escape from emergency escape locks at depths as great as one hundred feet (the record for successful escape is actually an astonishing 600 feet, representing a pressure of 300 psi). Loss of eardrums can occur, but this is not a life threatening injury.

The danger from overpressure comes from the collapse of buildings that are generally not as resistant. The violent implosion of windows and walls creates a hail of deadly missiles, and the collapse of the structure above can crush or suffocate those caught inside.

The dynamic pressure causes can cause injury by hurling large numbers of objects at high speed. Urban areas contain many objects that can become airborne, and the destruction of buildings generates many more. Serious injury or death can also occur from impact after being thrown through the air.

Blast effects are most dangerous in built-up areas due to the large amounts of projectiles created, and the presence of obstacles to be hurled against.

The blast also magnifies thermal radiation burn injuries by tearing away severely burned skin. This creates raw open wounds that readily become infected.

Radiation Injury

Ionizing radiation produces injury primarily through damage to the chromosomes. Since genetic material makes up a very small portion of the mass of a cell, the damage rarely occurs from the direct impact of ionizing radiation on a genetic molecule. Instead the damage is caused by the radiation breaking up other molecules and forming chemically reactive free radicals or unstable compounds. These reactive chemical species then damage DNA and disrupt cellular chemistry in other ways — producing immediate effects on active metabolic and replication processes, and long-term effects by latent damage to the genetic structure.

Cells are capable of repairing a great deal of genetic damage, but the repairs take time and the repair machinery can be overwhelmed by rapid repeated injuries. If a cell attempts to divide before sufficient repair has occurred, the cell division will fail and both cells will die. As a consequence, the tissues that are most sensitive to radiation injury are ones that are undergoing rapid division. Another result is that the effects of radiation injury depend partly on the rate of exposure. Repair mechanisms can largely offset radiation exposures that occur over a period of time. Rapid exposure to a sufficiently large radiation dose can thus cause acute radiation sickness, while a longer exposure to the same dose might cause none.

By far the most sensitive are bone marrow and lymphatic tissues — the

blood and immune system forming organs of the body. Red blood cells, which provide oxygen to the body, and white blood cells, which provide immunity to infection, only last a few weeks or months in the body and so must be continually replaced. The gastrointestinal system is also sensitive, since the lining of the digestive tract undergoes constant replacement. Although they are not critical for health, hair follicles also undergo continual cell division resulting in radiation sickness' most famous symptom — hair loss. The tissues least sensitive to radiation are those that never undergo cell division (i.e. the nervous system).

This also means that children and infants are more sensitive to injury than adults, and that fetuses are most sensitive of all.

If the individual survives, most chromosome damage is eventually repaired and the symptoms of radiation illness disappear. The repair is not perfect however. Latent defects can show up years or decades later in their effects on reproductive cells, and in the form of cancer. These latent injuries are a very serious concern and can shorten life by many years. They are the sole form of harm from low level radiation exposure.

Units of Measurement for Radiation Exposure

Three units of measurement have been commonly used for expressing radiation exposure: roentgens (R), rads, rems, the «three r's» of radiation measurement. In the scientific literature these are dropping out of use in favor of the SI (System Internationale) units grays (Gy) and sieverts (Sv). Each of the «three r's» measures something different. A rad is a measure of the amount of ionizing . A roentgen measures the amount of ionizing energy, in the form of energetic photons (gamma rays and x-rays) energy to which an organism is exposed. This unit is the oldest of the three and is defined more the convenience of radiation measurement, than for interpreting the effects of radiation on living organisms. Of more interest is the rad, since it includes all forms of ionizing radiation, and in addition measures the dose that is *actually absorbed* by the organism. A rad is defined as the absorption of 100 ergs per gram of tissue (or 0.01 J/kg). The gray measures absorbed doses as well, one gray equals 100 rads. The rem is also concerned with all absorbed ionizing radiations, and also takes into account the *relative effect* that different types of radiation produce. The measure of effect for a given radiation is its Radiation Biological Effect (RBE). A rem dose is calculated by multiplying the dose in rads for each type of radiation by the appropriate RBE, then adding them all up. The sievert is similar to the rem, but is derived from the gray instead of the rad. Sieverts use a somewhat simplified system of measuring biological potency — the quality factor (Q). One sievert is roughly equal to 100 rems. The rem and the sievert are the most meaningful unit for measuring and discussing the effects of radiation injury.

Acute Radiation Sickness

This results from exposure to a large radiation dose to the whole body within a short period of time (no more than a few weeks). There is no sharp cutoff to distinguish acute exposures from chronic (extended) ones. In general, higher total doses are required to produce a given level of acute sickness for longer exposure times. Exposures received over a few days do not differ substantially from instantaneous ones, except that the onset of symptoms is correspondingly delayed or stretched out. Nuclear weapons can cause acute radiation sickness either from prompt exposure at the time of detonation, or from the intense radiation emitted by early fallout in the first few days afterward.

The effects of increasing exposures are described below. A notable characteristic of increasing doses is the non-linear nature of the effects. That is to say, a threshold exists below which observable effects are slight and reversible (about 300 rems), but as exposures rise above this level the possibility of mortality (death) begins and increases rapidly with dose. This is believed to be due in part to the saturation of cellular repair mechanisms.

The total energy absorbed by a 75 kg individual with a whole body exposure of 600 rads (fatal in most cases) is 450 joules. It is interesting to compare this to the kinetic energy of a .45 caliber bullet, which is about 900 joules.

A power law for scaling radiation effects for longer term exposures has been proposed in which the dose required for a given effect increases by t^0.26, where time is in weeks. For exposures of one week or less the effect of rem of radiation is assumed to be constant. Thus an exposure capable of causing 50 % mortality is 450 rems if absorbed in a week or less, but is 1260 rems if it occurs over a year.

Acute Whole Body Exposure Effects

Below 100 REMS

In this dose range no obvious sickness occurs. Detectable changes in blood cells begin to occur at 25 rems, but occur consistently only above 50 rems. These changes involve fluctuations in the overall white blood cell count (with drops in lymphocytes), drops in platelet counts, and less severe drops in red blood cell counts. These changes set in over a period of days and may require months to disappear. They are detectable only by lab tests. At 50 rems atrophy of lymph glands becomes noticeable. Impairment to the immune system could increase the susceptibility to disease. Depression of sperm production becomes noticeable at 20 rems, an exposure of 80 rems has a 50 % chance of causing temporary sterility in males.

100–200 REMS

Mild acute symptoms occur in this range. Tissues primarily affected are the hematopoietic (blood forming) tissues, sperm forming tissues are also vulnera-

ble. Symptoms begin to appear at 100 rems, and become common at 200 rems. Typical effects are mild to moderate nausea (50% probability at 200 rems) , with occasional vomiting, setting in within 3-6 hours after exposure, and lasting several hours to a day. This is followed by a latent period during which symptoms disappear. Blood changes set in and increase steadily during the latency period as blood cells die naturally and are not replaced. Mild clinical symptoms return in 10–14 days. These symptoms include loss of appetite (50 % probability at 150 rems), malaise, and fatigue (50 % probability at 200 rems), and last up to 4 weeks. Recovery from other injuries is impaired and there is enhanced risk of infection. Temporary male sterility is universal. The higher the dosage in this range, the more likely the effects, the faster symptoms appear, the shorter the latency period, and the longer the duration of illness.

200–400 REMS

Illness becomes increasingly severe, and significant mortality sets in. Hematopoietic tissues are still the major affected organ system. Nausea becomes universal (100 % at 300 rems), the incidence of vomiting reaches 50 % at 280 rems. The onset of initial symptoms occurs within 1–6 hours, and last 1–2 days. After this a 7–14 day latency period sets in. When symptoms recur, the may include epilation (hair loss, 50 % probability at 300 rems), malaise, fatigue, diarrhea (50 % prob. at 350 rems), and hemorrhage (uncontrolled bleeding) of the mouth, subcutaneous tissue and kidney (50 % prob. at 400 rems). Suppression of white blood cells is severe, susceptibility to infection becomes serious. At 300 rems the mortality rate without medical treatment becomes substantial (about 10%). The possibility of permanent sterility in females begins to appear. Recovery takes 1 to several months.

400–600 REMS

Mortality rises steeply in this dose range, from around 50 % at 450 rems to 90 % at 600 (unless heroic medical intervention takes place). Hematopoietic tissues remain the major affected organ system. Initial symptoms appear in 0.5–2 hours, and last up to 2 days. The latency period remains 7-14 days. The symptoms listed for 200–400 rems increase in prevalence and severity, reaching 100 % occurrence at 600 rems. When death occurs, it is usually 2–12 weeks after exposure and results from infection and hemorrhage. Recovery takes several months to a year, blood cell counts may take even longer to return to normal. Female sterility becomes probable.

600–1000 REMS

Survival depends on stringent medical intervention. Bone marrow is nearly or completely destroyed, requiring marrow transfusions. Gastrointestinal tissues are increasingly affected. Onset of initial symptoms is 15–30 minutes, last a day or two, and are followed by a latency period of 5–10 days. The final phase lasts 1 to 4 weeks, ending in death from infection and internal bleeding. Recovery, if

it occurs, takes years and may never be complete.

Above 1000 REMS

Very high exposures can sufficient metabolic disruption to cause immediate symptoms. Above 1000 rems rapid cell death in the gastrointestinal system causes severe diarrhea, intestinal bleeding, and loss of fluids, and disturbance of electrolyte balance. These effects can cause death within hours of onset from circulatory collapse. Immediate nausea occurs due to direct activation of the chemoreceptive nausea center in the brain.

In the range 1000–5000 rems the onset time drops from 30 minutes to 5 minutes. Following an initial bout of severe nausea and weakness, a period of apparent well-being lasting a few hours to a few days may follow (called the «walking ghost» phase). This is followed by the terminal phase which lasts 2–10 days. In rapid succession prostration, diarrhea, anorexia, and fever follow. Death is certain, often preceded by delirium and coma. Therapy is only to relieve suffering.

Above 5000 rems metabolic disruption is severe enough to interfere with the nervous system. Immediate disorientation and coma will result, onset is within seconds to minutes. Convulsions occur which may be controlled with sedation. Victim may linger for up to 48 hours before dying.

The U.S. military assumes that 8000 rads of fast neutron radiation (from a neutron bomb) will immediately and permanently incapacitate a soldier.

It should be noted that people exposed to radiation doses in the 400-1000 rem range following the Chernobyl disaster had much higher rates of survival than indicated above. This was made possible by advances in bone marrow transfusions and intensive medical care, provided in part by Dr. Robert Gale. However two caveats apply:

Such care is only available if the number of cases is relatively small, and the infrastructure for providing it is not disrupted. In the case of even a limited nuclear attack it would be impossible to provide more than basic first aid to most people and the fatality rates might actually be higher than given here.

Many of the highly exposed Chernobyl survivors have since died from latent radiation effects.

Acute Localized Tissue Exposure

Localized acute exposure is important for two organs: the skin, and the thyroid gland.

Beta Burns

Beta particles have a limited range in tissue. Depending on their energy, betas are completely absorbed by 1 mm to 1 cm of tissue. External exposures to beta particles from fallout thus primarily affect the skin, causing «beta burns». Due

to the poor penetrating power of betas, these injuries only occur if there is direct skin exposure to fallout particles, or if an individual remains outdoors in a strong radiation field. Remaining indoors, wearing substantial clothing, and decontamination by washing can prevent this type of exposure. Beta burns were encountered in Marshall Islanders, and the crew of a Japanese fishing vessel, following the Castle Bravo test which unexpectedly dumped high fallout levels over a large area.

The initial symptom for beta burns are an itching or burning sensation during the first 24–48 hours. These symptoms are marked only if the exposure is intense, and do not occur reliably. Within 1-2 days all symptoms disappear, but after 2–3 weeks the burn symptoms appear. The first evidence is increased pigmentation, or possibly erythema (reddening). Epilation and skin lesions follow.

In mild to moderate cases damage is largely confined to the epidermis (outer skin layers). After forming a dry scab, the superficial lesions heal rapidly leaving a central depigmented area, surrounded by an irregular zone of increased pigmentation. Normal pigmentation returns over a few weeks.

In more serious cases deeper ulcerated lesions form. These lesions ooze before becoming covered with a hard dry scab. Healing occurs with routine first aid care. Normal pigmentation may take months to return.

Hair regrowth begins 9 weeks after exposure and is complete in 6 months.

Thyroid Exposure

The short-lived radioisotope iodine-131 (half-life 8 days) presents a special risk due to the tendency for ingested iodine to be concentrated in the thyroid gland. This risk is mitigated by the fact that direct ingestion of fallout is rare, and easily avoided. Iodine-131 typically enters the body through the consumption of contaminated milk, which in turn results from milk cows consuming contaminated fodder.

The short half-life means that the initial radiation intensity of I-131 is high, but it disappears quickly. If uncontaminated fodder can be provided for a month or two, or if dry or canned milk can be consumed for the same period, there is little risk of exposure.

If I-131 contaminated food is consumed, about one-third of the ingested iodine is deposited in the thyroid gland which weighs some 20 g in adults, and 2 g in infants. This can result in very high dose rates to the gland, with negligible exposures to the rest of the body. Due to the smaller glands of infants and children, and their high dairy consumption, they are particularly vulnerable to thyroid injury. Some Marshallese children received thyroid doses as high as 1150 rems. Most of the children receiving doses over 500 rems developed thyroid abnormalities within 10 years, including hypothyroidism and malignancies.

I-131 exposure can be prevented by prompt consumption of potassium iodide supplements. Large doses of potassium iodide saturate the body with iodine and prevent any subsequent retention of radioiodine that is consumed.

Fetal Injury

Acute radiation exposure during pregnancy can cause significant harm to the fetus. At Hiroshima and Nagasaki adverse effects were seen when pregnant women who were exposed to 200 rems of radiation or more. When exposure occurred during the first trimester a significant increase in mentally impaired children were noted. When exposure occurred during the last trimester, there was a marked increase in stillbirths and in elevated infant mortality during the first year of life.

Chronic Radiation Exposure

Long term radiation exposure results from residing in a fallout contaminated area for an extended period (external exposure), consuming food produced in a contaminated area (internal exposure), or both. If the exposure rate is low enough, no symptoms of radiation sickness will appear even though a very large total radiation dose may be absorbed over time. Latent radiation effects (i.e. cancer, genetic damage) depend on total dosage, not dose rate, so serious effects can result. An exposure of 0.25 rem/day over 5 years would accumulate 450 rems with little chance of overt sickness, but it would have a high mortality rate if the exposure were acute.

The exposure time scaling law given above also indicates that a slow onset of symptoms characteristic of acute radiation sickness can occur. As an example, the most heavily contaminated location of the Rongelap atoll (160 km downwind of the March 1, 1954 15 Mt Castle Bravo test), received a total accumulated exposure of 3300 rads. Of this, 1100 rads was accumulated during the interval from 1 month to 1 year following the test. If the site had been occupied during this period, the effective exposure for radiation sickness effects would be 1100/(48 weeks)^0.26 = 403 rads.

External Exposure

When an area is contaminated by gamma emitting isotopes, a radiation field is created that exposes all organisms that are not shielded from it. Only gamma rays have the necessary range and penetration to create a significant hazard. The principal source of long-term external exposure is cesium-137 (30 year half-life, 0.6 MeV gamma energy).

A megaton of fission yield produces enough Cs-137 to contaminate 100 km^2 with a radiation field of 200 rad/year. A megaton-range ground burst can contaminate an area of thousands of square kilometers with concentrations that would exceed occupational safety guidelines. 3,000 megatons of fission yield, if distributed globally by stratospheric fallout, would double the world's background radiation level from external exposure to this isotope alone.

It is possible to substantially reduce external exposure in contaminated areas by remaining indoors as much as possible. Exposure can be reduced by a factor of 2–3 for a frame house, or 10–100 for a multi-story building, and adding additional shielding to areas where much time is spent (like the bedroom) can increase

these factors substantially. Since the half-life of Cs-137 is long, these would be permanent lifestyle adjustments. Such measures have been necessary (especially for children) in areas of Belarus that were heavily contaminated by Chernobyl.

Internal Exposure

Internal exposure to radiation is the most serious chronic risk from fallout if food grown in contaminated areas is consumed. Widespread contamination from a nuclear war, or a major radiation accident (like the Kyshtym and Chernobyl disasters), may leave no other practical choice. Alternatively, people residing in contaminated areas may come to disregard safety instructions about locally produced food (as has happened in the Marshall Islands and Ukraine).

Radioisotopes may be taken up into plants through the root system, or they may be contaminated by fallout descending on the leaves. Gross contamination of food plants or fodder from the fallout plume of a ground burst is an obvious hazard, but the gradual descent of worldwide fallout is also a problem.

The primary risks for internal exposure are cesium-137 and strontium-90. Strontium-89, transuranics alpha emitters, and carbon-14 are also significant sources of concern.

Only a few curies of radioisotopes per km^2 are sufficient to render land unsuitable for cultivation under current radiation safety standards. A megaton of fission yield can thus make some 200,000 km^2 useless for food production for decades. Depression of leukocyte levels have been observed in people in Belarus living in areas that were contaminated with only 0.2 curies/km^2.

Cesium-137

This alkali metal has chemistry resembling that of potassium. As a result, it is readily absorbed by food plants, and by animal tissues. Once consumed cesium distributes itself fairly evenly through the body, which means that Cs-137 absorption causes whole body exposure (a fact further aided by the penetrating nature of its gamma emissions). Cesium has a moderate residence time in the body, the residence half-life ranging from 50–100 days, so that the body will be cleared of the isotope once consumption of contaminated material ceases in a matter of several months, to a few years.

Strontium 90 and 89

Strontium is chemically similar to calcium, and is deposited in bone along with calcium. Most of the strontium ingested does not end up in bone, it has a biological half-life only 40 days. Somewhat less than 10 % of the Sr is retained in the bone, but it has a biological half-life of 50 years. Since the bone marrow is among the most sensitive tissue in the body to radiation, this creates a very serious hazard.

Sr-90 (28.1 yr half-life) thus can cause long term damage, while Sr-89 (52 days) can cause significant short term injury. Safety exposure standards im-

pose a Sr-90 body burden limit of 2 microcuries (14 nanograms) for occupational exposure, 0.2 microcuries for individual members of the general population, and 0.067 microCi averaged over the whole population. It is estimated that 10 microCi per person would cause a substantial rise in the incidence of bone cancer. The explosion of several thousands of fission megatons in the atmosphere could raise the average body burden of the entire human race to above the occupational exposure limit for Sr-90 for a couple of generations. Contamination of 2 curies of Sr-90 per km^2 is the U.S. limit for food cultivation.

Alpha emitting heavy elements can be serious health risks also. The isotopes of primary concern here are those present in substantial quantities in nuclear weapons: short lived uranium isotopes (U-232 and U-233) and transuranic elements (primarily Pu-239, Pu-240, and Americium-241). These elements are hazardous if ingested due to radiotoxicity from the highly damaging alpha particles. The quantities of these isotopes present after a nuclear explosion are negligible compared to the amount of fission product radioisotopes. They represent a hazard when nuclear weapons are involved in «broken arrow» incidents, that is, accidents where the fissile isotopes inside are released. The exposure areas are of course small, compared to the areas threatened by fallout from a nuclear detonation. A typical nuclear weapon will contain some 300–600 curies of alpha emitter (assuming 5 kg plutonium). The isotope breakdown is approximately: 300 curies Pu-239, 60 curies Pu-240, and up to 250 curies of Am-241.

If small particles of alpha emitters are inhaled, they can take up permanent residence in the lung and form a serious source of radiation exposure to the lung tissue. A microcurie of alpha emitter deposited in the lungs produce an exposure of 3700 rems/yr to lung tissue, an extremely serious cancer risk.

Uranium and the transuranic elements are all bone-seekers (with the exception of neptunium). If absorbed, they are deposited in the bone and present a serious exposure risk to bone tissue and marrow. Plutonium has a biological half-life of 80–100 years when deposited in bone, it is also concentrated in the liver with a biological half-life of 40 years. The maximum permissible occupational body burden for plutonium-239 is 0.6 micrograms (.0375 microcuries) and 0.26 micrograms for lung burden (0.016 microCi).

Carbon-14 is a weak beta particle emitter, with a low level of activity due to its long half-life. It presents a unique hazard however since, unlike other isotopes, it is incorporated directly into genetic material as a permanent part throughout the body. This means that it presents a hazard out of proportion to the received radiation dose as normally calculated.

Cancer

The most serious long term consequence of radiation exposure is the elevation of cancer risk. Estimates of the carcinogenicity of radiation, especially of low exposures, have tended to increase over the years as epidemiological data

has accumulated.

The current state-of-the-art in low level risk estimation is the 1990 report issued by the National Academy of Sciences Committee on Biological Effects of Ionizing Radiation (BEIR) entitled _Health Effects of Exposure to Low Levels of Ionizing Radiation_, also known as BEIR V.

As a general rule of thumb, it appears that cancer risk is more or less proportional to total radiation exposure, regardless of the quantity, rate or duration. 500 rems received over a decade is thus as serious a risk as 500 rems received all at once, and 50 rems is one-tenth as bad as 500. There is no evidence of a threshold effect or «safe dose». Safety standards are established primarily to keep the increased incidence of cancer below detectable levels.

Significant deviations from the above rule of proportionality for total exposure do occur. In particular, low doses (for which the risk is small anyway) received over an extended period of time are significantly less carcinogenic (by about a factor of 2) than the same dose received all at once.

Cancer risk to radiation exposure can be expressed as the increase in the lifetime probability of contracting fatal cancer per unit of radiation. The current estimate of overall risk is about a 0.8 % chance of cancer per 10 rems for both men and women, averaged over the age distribution of the U.S. population. Thus a 1000 rem lifetime whole body radiation exposure would bring about a 80 % chance of contracting fatal cancer, in addition to the normal incidence of cancer (about 20 %). The risk for children appears to be about twice as great (due at least partly to the fact that they will live longer after exposure, and thus have greater opportunity to contract cancer).

Genetic Effects

Radiation damage to the germ cells of the reproductive organs can cause mutations that are passed on to subsequent generations. Although this is very important, it can nonetheless be overplayed. It may seem surprising, but no elevated mutation rate from radiation has ever been detected in the human population, not even in the substantial population of atomic bomb survivors and descendants. One reason for this is that humans are wild animals, that is, they have not been subjected to controlled breeding and thus have a high incidence of natural genetic variability and disorders, compared to laboratory and domestic animals. About 10 % of the human population has detectable genetic disorders (most are not serious). This makes it difficult to detect additional mutations unless the rate is also high.

Two factors act to limit the effective radiation exposure for genetic effects, one for acute exposures, the other for chronic exposures. High acute exposures to the reproductive organs can cause permanent sterility, which prevents transmission of genetic effects. The cumulative effect of chronic exposure is limited by the fact that only exposures prior to reproduction count. Since most reproduc-

tion occurs before the age of 30, exposures after that age have little effect on the population.

It is estimated that the dose to reproductive tissue required to double the natural incidence of genetic disorders is 100–200 rems. The initial rate of observable disorders (the first generation) is only about 1/3 of the eventual rate once genetic equilibrium is established. Of course increases in the rate of genetic disorders (especially in a large population) is a _permanent_ alteration of the human species.

Cataracts

Eye tissue exposed to radiation shows an increased incidence of cataracts at dose levels below which most tissues show increased cancer rates. This makes cataract risk the most important tissue dose criterion for establishing safety standards.

CHAPTER 3
CHEMICAL DETECTION TECHNOLOGIES

The applicability of chemical detection equipment to potential user groups will be dependent upon the characteristics of the detection equipment, as well as the type of CA and TIC/TIM detected and the objective of the first responder unit. Numerous technologies are available for the detection of CA and TIC/TIM vapors; some technologies are available for detection and identification of liquid droplets of CAs on surfaces; and many laboratory-based technologies exist for detection of TICs/TIMs in water. The quality of analytical results from the various analyzers is dependent upon the ability to effectively sample the environment and get the sample to the analyzer.

Equipment designed for vapor detection will not be readily applicable for detection of low volatility liquid contamination on surfaces or contamination in water. In addition, vapor detection equipment could have difficulty in identifying a small amount of CA or TIC/TIM in a high background of nonhazardous environmental chemicals. For example, a chemical vapor detector may readily detect trace levels of CAs or TICs/TIMs in a rural setting such as a forest or an open field, but the same detector may not be capable of detecting the same level of CA or TIC/TIM in an urban setting such as a crowded subway station or busy city street. More urban environments typically contain many chemicals produced by everyday human activities (driving an automobile, deodorant/perfumes use, insecticide/herbicide application, etc.) that look like a CA or TIC/TIM to the detection equipment and may affect the reliability (number of false readings) of the instrument as well as its sensitivity. However, by testing the equipment prior to an emergency use, the operator can become familiar with the idiosyncrasies of the detection equipment when exposed to various environmental chemicals expected in operational areas. As technological advances continue to be made, more effective and accurate methods of detection that are less affected

by environmental chemicals in operational areas will become commercially available at lower costs.

Chemical agents can be detected by several means that incorporate various technologies. The technologies discussed in this guide are grouped into three major categories: point detection, standoff detection, and analytical instruments. The technology needed for CA and TIC/TIM detection will be dependent on the CA or TIC/TIM used and the objective of the first responder unit.

Point Detection Technologies

Point detection technology is applicable in determining the presence of CA or TIC/TIM and can be used to map out contaminated areas if enough time is available. Point detectors can be used as warning devices to alert personnel to the presence of a toxic vapor cloud. In this scenario, the detector is placed up-wind of the first responder location. When the toxic chemical is carried towards this location, it first encounters the detector, thus sounding an alarm and allowing the first responders to don the necessary protective clothing. It should be noted that if the concentration of CA or TIC/TIM is high enough to be immediately life threatening, point detectors may not provide sufficient time to take protective measures.

Another use of a point detector would be to monitor the vapor contamination originating from a decontamination site. Point detectors can also be used during post-release triage to determine the contamination level of each person (i.e., highly contaminated personnel, lightly contaminated personnel, and uncontaminated personnel) with the idea that all contaminated people need rapid decontamination while noncontaminated people do not need to be decontaminated. This allows for conservation of decontamination resources and prevents wasted effort on noncontaminated personnel. The following point detection techniques were identified:

- Ionization/Ion Mobility Spectrometry.
- Flame Photometry.
- Infrared Spectroscopy.
- Electrochemistry.
- Colorimetric.
- Surface Acoustic Wave.
- Photoionization Detection.
- Thermal and Electrical Conductivity.
- Flame Ionization.
- Polymer Composite Detection Materials.

Ionization/Ion Mobility Spectrometry

A detector using ionization/ion mobility spectrometry (IMS) technology is typically a stand-alone detector that samples the environment using an air pump. Contaminants in the sampled air are ionized by a radioactive source, and the result-

ant ions traverse the drift tube through an electric field toward an ion detector. The flight time, or the time it takes the ions to traverse the distance, is proportional to the size and shape of the ionized chemical species and is used for identification of the species. Analysis time ranges from several seconds to a few minutes.

Ionization of gaseous species can be achieved at atmospheric pressure. Using proton transfer reactions, charge transfer, dissociative charge transfer, or negative ion reactions such as ion transfer, nearly all chemical classes can be ionized. However, most IMS portable detectors use radioactive electron (beta ray) emitters to ionize the sample.

Because IMS requires a vapor or gas sample for analysis, liquid samples must first be volatilized. The gaseous sample is drawn into a reaction chamber by a pump where a radioactive source, generally ^{63}Ni (Nickel-63) or Am (Americium-241), ionizes the molecules present in the sample. The ionized air sample, including any ionized CA, is then injected into a closed drift tube through a shutter that isolates the contents of the drift tube from the atmospheric air. The drift tube has an electrical charge gradient that draws the sample towards a receiving electrode at the end of the drift tube. Upon ion impact, an electrical charge is generated and recorded with respect to a travel time. The travel time is measured from the opening of the shutter to the signal appearance at the receiving electrode. The ions impact the electrode at different intervals providing a series of peaks and valleys in electrical charge that is usually graphed on Cartesian Coordinates. The Y-axis corresponds to the intensity of the charge received by impact of the various species that have respective travel times in the drift tube. This travel time in the drift tube and the strength of the charge gives a relative concentration of species in the sample. An example of a handheld detector using IMS technology is the Advanced Portable Detector (APD), manufactured by Smiths Detection. This detector is shown in figure 1.

Figure 1 — Advanced Portable Detector (APD) 2000, Smiths Detection

The M8A1 Automatic Chemical Agent Alarm System is another example of an IMS technology CA detection and warning system. It incorporates the M43A1 detector to detect the presence of nerve agent vapors or inhalable aerosols. The M43A1 detector is an ionization product diffusion/ion mobility type detector. Air is continuously drawn through the internal sensor by a pump at a

rate of approximately 1.2 L/min. Air and agent molecules are first drawn past a radioactive source (^{241}Am) and a small percentage are ionized by the radiation. The air and agent ions are then drawn through the baffle sections of the cell. The lighter air ions diffuse to the walls and are neutralized more quickly than the heavier agent ions that have more momentum and are able to pass through the baffled section. As a result, the collector senses a greater ion current when nerve agents are present compared to the current when only clean air is sampled. An electronic module monitors the current produced by the sensor and triggers the alarm when a critical threshold of current is reached.

Differential ion mobility spectrometry (DMS) is one more example of an IMS technology for detection and identification of analytes in a volatilized sample. DMS separates ions by measuring the difference between ion mobilities as they pass through applied electrical fields.

Flame Photometry

Flame photometry is based on burning ambient air with hydrogen gas. The flame decomposes any CAs or TIMs present in the air, and the characteristic radiation emitted by the particular excited molecular species during its transition to the ground state can be measured. Sulfur- and phosphorous-containing compounds introduced in a hydrogen-rich flame decompose, giving rise to excited S_2* and HPO* molecular species respectively, where * represents the excited atomic or molecular state. At the elevated flame temperature, the phosphorus and sulfur emit light of specific wavelengths. These chemiluminescent emissions are isolated by appropriate narrow band optical filters and converted into measurable electrical signals by a photomultiplier tube, which produces an analog signal related to the concentration of the phosphorus- and sulfur-containing compounds in the air. Since the classical nerve agents all contain phosphorus and sulfur and mustard contains sulfur, these agents are readily detected by flame photometry. Flame photometry is sensitive and allows ambient air to be sampled directly. However, it is also prone to false alarms from interferents that contain phosphorus and sulfur. The number of false positives due to interference can be minimized using algorithms. Using a flame photometric detector (FPD) in cooperation with a gas chromatograph will further reduce the likelihood of false alarms. There are a number of gas chromatographs that use FPDs for detection purposes.

An example of a handheld detector using this technology is the APACC Chemical Control Alarm Portable Apparatus, manufactured by Proengin SA. This detector is shown in figure 2.

Figure 2 — APACC Chemical Control Alarm Portable Apparatus, Proengin SA

Infrared Spectroscopy

Infrared (IR) spectroscopy is the measurement of the wavelength and intensity of the absorption of mid-infrared light by a sample. Mid-infrared light, bandwidth (2.5 |im to 50 |im) and frequency (4000 cm^{-1} to 200 cm^{-1}), is energetic enough to excite molecular vibrations to higher energy levels. The wavelengths of IR absorption bands are characteristic of specific types of chemical bonds and every molecule has a unique IR spectrum (fingerprint). Infrared spectroscopy finds its greatest utility for identification of organic and organometallic molecules. There are two IR spectroscopy technologies employed in point detectors: photoacoustic infrared spectroscopy (PIRS) and filter-based IR spectroscopy. These two technologies and specific detector examples are discussed in the remainder of this section.

Photoacoustic Infrared Spectroscopy

Photoacoustic infrared spectroscopy (PIRS) detectors use the photoacoustic effect to identify and detect CA vapors. Infrared radiation is pulsed into a sample that selectively absorbs specific IR wavelengths characteristic of target gases. When the gas absorbs IR radiation, its temperature rises, which causes the gas to expand and produces an acoustical wave that can be detected by microphones mounted inside the sample cell. Various filters are then used to selectively transmit specific IR wavelengths absorbed by the CA being monitored. Selectivity can be increased by sequentially exposing the sample to several wavelengths of light. Using multiple wavelengths to identify the unknown decreases the chance of contaminants that cause false positives and fewer interferents will be observed. Chemical agents are distinguished from interferents by the relative signal produced when several different wavelengths are sequentially transmitted to the sample.

When CA is present in the sample, an audible signal (at the frequency of modulation) is produced by the absorption of the modulated IR light. Quantitation is possible because the acoustical wave is directly proportional to the concentration of the gas inside the cell. Although photoacoustic detectors are sensitive to external vibration and humidity, as long as the detector is calibrated in each operating environment immediately prior to sampling, selectivity will be very high. One mobile laboratory unit that utilizes photoacoustic IR spectrosco-

py technology is the Innova Type 1412 Multigas Monitor, from California Analytical Instruments, shown in figure 3.

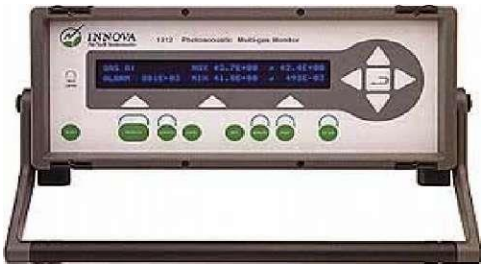

Figure 3 — Innova Type 1412 Multigas Monitor, California Analytical Instruments

Filter-Based Infrared Spectrometry

Filter-based infrared spectrometry is based on a series of lenses and mirrors that directs a narrow bandpass IR beam in a preselected path through the sample. The amount of energy absorbed by the sample is measured and stored in memory. The same sample is examined at as many as four additional wavelengths. This multiwavelength, multicomponent data is analyzed by the microprocessor utilizing linear matrix algebra. Concentrations of each component, in each sample, at each station, are used for compiling time weighted average (TWA) reports and trend displays. The data management and control software (DMCS) retains data for further analysis and longer term storage and retrieval. Thermo Fisher Scientific produces a portable ambient air analyzer, the Miran SaphIRe Portable Ambient Air Analyzer that is shown in figure 4.

Figure 4 — Miran SaphIRe Portable Ambient Air Analyzer, Thermo Fisher Scientific

Electrochemistry

Electrochemical detectors monitor the resistance of a thin film that changes as the film absorbs chemicals from the air or monitors a change in the electric potential of an electrode when chemicals in solution or in air are absorbed. Although electrochemical detectors are selective, they are not as sensitive as technologies such as IMS and flame photometry. Hot and cold temperatures change the rates of reactions and shift the equilibrium point of the various reactions, which affects sensitivity and selectivity. Several of the fielded electrochemical detectors encounter problems when exposed to environmental extremes.

The inhibition of cholinesterase by nerve agents is an example of one type of reaction that can be detected by this technique. A solution containing a known amount of cholinesterase is exposed to an air sample that may contain nerve agent. If nerve agent is present, a percentage of the cholinesterase will be inhibited from reaction in the next step, that is, the addition of a solution containing a compound that will react with uninhibited cholinesterase to produce an electrochemically active product. The resulting cell potential is related to the concentration of uninhibited cholinesterase, which is related to the concentration of nerve agent present in the sampled air. Another type of electrochemical detector monitors the resistance of a thin film that increases as the film absorbs CA from the air. An example of a handheld detector using this technology is the ToxiRAE Plus Personal Gas Monitor manufactured by RAE Systems, Inc. (figure 5).

Figure 5 — ToxiRAE Plus Personal Gas Monitor, RAE Systems, Inc

Colorimetric

Colorimetric chemistry is a wet chemistry technique formulated to indicate the presence of a CA by a chemical reaction that causes a color change when agents come in contact with certain solutions or substrates. The color change can be detected either visibly or with spectrophotometric devices. Detection tubes, papers, or tickets are common and can be used to detect nerve, blister, and blood agents. Detection paper is the least expensive and sophisticated technique for detection and can be used to quickly detect liquids and aerosols when defining a contaminated area, but it lacks specificity and can result in false-positive determinations with common chemicals such as antifreeze, brake fluid, or insect repellant. Normally, two dyes and one pH indicator are used, which are mixed with cellulose fibers in a paper without special coloring (unbleached). When a drop of chemical warfare agent is absorbed by the paper, it dissolves one of the pigments. Mustard agent dis-

solves a red dye and nerve agent a yellow. In addition, VX causes the indicator to turn blue that, together with the yellow, will become green/green-black.

Detector papers are generally used for testing suspect droplets or liquids on a surface. For gaseous or vaporous CAs, colorimetric tubes are available. The colorimetric tubes consist of a glass tube that has the reacting compound sealed inside. Upon use, the tips of the tubes are broken off and a pump is used to draw the sample across the reacting compound (through the tube). If a CA is present, a reaction resulting in a color change takes place in the tube. Colorimetric tubes are typically used for qualitative determinations, to verify the presence of a CA after an alarm is received from another monitor. They can also be used to test drinking water for contamination. Draeger Safety, Inc., manufactures a number of colorimetric tubes. A picture of the Draeger CDS Kit is shown in figure 6.

Figure 6 — Draeger CDS Kit, Draeger Safety, Inc.

Surface Acoustic Wave

Surface acoustic wave (SAW) detectors consist of piezoelectric crystals coated with a film designed to absorb CAs from the air. The SAW sensors detect changes in the properties of acoustic waves as they travel at ultrasonic frequencies in the piezoelectric materials. Target gases are absorbed onto chemically selective surfaces, which cause a change in the resonant frequency of the piezoelectric crystal. The SAW detectors use two to six piezoelectric crystals that are coated with different polymeric films. Each polymeric film preferentially absorbs a particular class of volatile compound. For example, one polymeric film will be designed to preferentially absorb water, while other polymer films are designed to preferentially absorb different types of chemicals such as trichloroethylene, toluene, ethyl-benzene, or formaldehyde. The piezoelectric crystals detect the mass of the chemical vapors absorbed into the different, chemically selective polymeric coatings. The change in mass of the polymeric coatings causes the resonant frequency of the piezoelectric crystal to change. By monitoring the resonant frequency of the different piezoelectric crystals, a response pattern of the system for a particular vapor is generated. This response pattern is then stored in a microprocessor. When the system is operating, it constantly compares each new response pattern to the stored response pattern for the target vapor. When the response pat-

tern for the target vapor matches the stored pattern, the system alarm is activated.

Arrays of these sensors are used to simultaneously identify and measure many different CAs. A preconcentration tube can be used to further increase detection sensitivity. These relatively inexpensive devices can be handheld and have several advantages, including rapid response (about 2 s), 100 % reversible recovery in 5 s to 100 s, parts per trillion (ppt) sensitivity in quantitative determinations, and a long lifetime (>1 yr) for the polymer coatings. The selectivity and sensitivity of these detectors depends on the ability of the film to absorb only the suspect CAs from the sample air. Operation is simple and involves very little training or expertise. Many SAW devices use preconcentration tubes to reduce environmental interferences and increase the detection sensitivity. A detector manufactured by Microsensor Systems, Inc., that is based upon the SAW technology is the SAW MiniCAD mkII (figure 7).

Figure 7 — SAW MiniCAD mkII, Microsensor Systems, Inc.

Photoionization Detection

Photoionization detection (PID) works by exposing a gas stream to an ultraviolet light of a wavelength with enough energy to ionize an agent molecule. If agents are present in the gas stream, they are ionized, and an ion detector then registers a voltage proportional to the number of ions produced in the gas sample, which is the concentration of the agent. Specificity of these detectors is a function of how narrow the spectral range of the exciting radiation is and on how unique that energy is to ionizing only the molecule of interest. RAE Systems, Inc., produces the MiniRAE 2000, a handheld detector that utilizes the PID technology, shown in figure 8.

Figure 8 — MiniRAE 2000, RAE Systems, Inc.

Thermal and Electrical Conductivity

Thermal and electrical conductivity detectors use metal oxide thermal semiconductors that measure the change in heat conductivity that occurs as a result of gas adsorption on the metal oxide surface. In addition, the change in resistance and electrical conductivity across a metal foil in the system is measured when a gas adsorbs onto the surface of the metal film. Contaminants in the atmosphere being measured will result in measurable electrical differences from the «clean» or background atmosphere. However, since different contaminants will have different thermal conductivities and, therefore, different electrical responses from the detector, this technology is relatively nonselective. An example of a handheld detector using this technology is the Portable Odor Monitor, manufactured by Sensidyne, Inc., (figure 9).

Figure 9 — Portable Odor Monitor, Sensidyne, Inc.

Flame Ionization

A flame ionization detector (FID) is a general-purpose detector used to determine the presence of volatile carbon-based compounds that are incinerated in a hydrogen-oxygen or hydrogen-air flame. When the carbonaceous compounds burn, ions are generated that cause an increase in the flame's baseline ion current at a collection electrode in proximity to the flame. The FIDs are not specific and require separation technology for specificity, such as a gas chromatograph. Identification of compounds is generally determined by comparison of the chromatographic retention time of a compound to that of a known standard, or to chromatographic retention indices for a series of known compounds using a standard set of chromatographic conditions. Thermo Fisher Scientific manufactures a unit, the TVA-1000B (FID or FID/PID) Toxic Vapor Analyzer for the specific determination of GA at 0.61 ppm (v) (above IDLH) and HD at 0.29 ppm (v) (no IDLH). The TVA-1000B is shown in figure 10.

Figure 10 — TVA-1000B (FID or FID/PID) Toxic Vapor Analyzer, Thermo Fisher Scientific

Polymer Composite Detection Materials

Polymer composite detection materials consist of individual thin-film carbon-black/polymer composite chemi-resistors configured into an array. The detection materials are deposited as thin films on an alumina substrate across two electrical leads, creating conducting chemi-resistors.

The output from the device is an array of resistance values measured between each of the two electrical leads for each of the detectors in the array. Nerve agent simulants, such as dimethylmethylphosphonate (DMMP) and diisopropylmethylphosponate (DIMP), could be resolved from test analytes, including water, methanol, benzene, toluene, diesel fuel, lighter fluid, vinegar, and tetrahydrofuran, by using standard data analysis techniques to assess the collective output of the array. The Cyranose® 320, from Smiths Detection, pictured in fig. 11, is a polymer composite detection materials device.

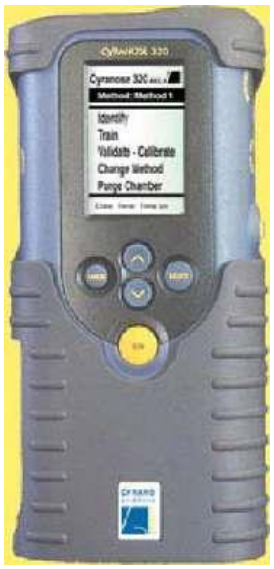

Figure 11 — Cyranose® 320, Smiths Detection

Standoff Detectors

Standoff detectors are used to give advance warning of a CA cloud. Standoff detectors typically use optical spectroscopy and can detect CAs at distances as great as 5 km. Agent-free spectra are used as a baseline to compare with freshly measured spectra that may contain CA. Standoff detectors are generally difficult to operate and usually require the operator to have some knowledge of spectroscopy in order to interpret results. Passive standoff detectors collect IR radiation emitted and/or measure IR radiation absorbed from the background to detect CA and TIM vapor clouds. The following standoff techniques were identified:

- Fourier Transform Infrared and Forward Looking Infrared.
- Ultraviolet Standoff.

Fourier Transform Infrared and Forward Looking Infrared

Fourier transform infrared (FTIR) and forward looking infrared (FLIR) spectrometers remotely monitor an area by either collecting IR radiation emitted or measuring IR radiation absorbed from the background to detect CA and TIM vapor clouds. In order to detect the various wavelengths emitted from the vapor clouds, FTIR spectroscopy uses an interferometer to process the IR radiation and FLIR spectroscopy uses a series of optical filters. Through the use of computer-based Fourier signal processing, rapid scan rates of wide ranges of wavelength and a spectrum with characteristic «fingerprint» peaks that can be used to identify the detected chemical can be generated. An example of a handheld detector using this technology is the HAWK Long Range Chemical Detector, manufactured by Bruker Daltonics, Inc. (figure 12). Another portable detector using this technology is the HazMatID from Smiths Detection, shown in figure 13.

Figure 12 — HAWK Long Range Chemical

Figure 13 — HazMatID, Smiths Detection Detector, Bruker Daltonics, Inc.

Ultraviolet Standoff

Certain compounds have the ability to absorb ultraviolet (UV) light. Characteristic UV absorptions can be useful in identifying species or assisting in determining structure. Ultraviolet spectroscopy equipment, such as the Safeye 400 Gas Detection System, manufactured by Spectrex, Inc. (figure 14), have several advantages, including direct fast response to changes in gas concentrations, capability of large area surveillance, good cost effectiveness, and ability to remain unaffected by environmental conditions such as heat, humidity, snow, or rain. Disadvantages of standoff detectors include the inability to indicate the precise concentration at a given point and dependence on an unobstructed line of sight between beam emitter and detector.

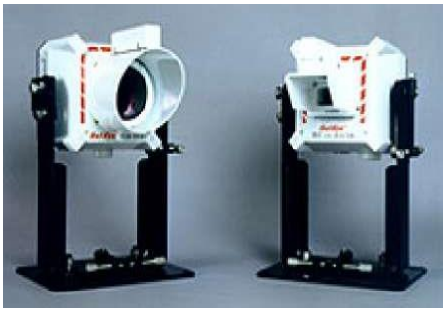

Figure 14 — Safeye Model 400 Gas Detection System (UV), Spectrex, Inc.

Analytical Instruments

The analytical instruments described in this section can be used to analyze samples as small as a few microliters or milligrams. They are designed to differentiate between and accurately measure the unique chemical properties of different molecules. Accuracy and reliability requires that only very pure reagents be used, very rigid protocol and operating procedures be followed, and careful handling be employed to prevent contamination and malfunction. Since the instruments do not display the measured data in a straightforward manner, interpretation of the measured data generally requires a technical background and extensive formal training. This typically precludes their use outside of a laboratory environment, which is staffed by technically trained people.

However, some analytical instruments have been developed for field applications. The following analytical techniques were identified.

- Gas Chromatography.
- Mass Spectrometry.
- High-Performance Liquid Chromatography.
- Ion Chromatography.
- Capillary Zone Electrophoresis.
- Ultraviolet Spectrometry.

Gas Chromatography

In Gas Chromatography (GC) applications, an inert gas (mobile phase) is used to transport a volatile multicomponent sample through a long chromatographic column (packed or coated with stationary phase) in order to separate analytes in a mixture from interferences for subsequent detection. As the sample flows through the column, the various components of the sample partition between the mobile and stationary phases at different rates depending on their chemical identity or affinity for the stationary phase. The time spent (retention time) for each component of a mixture to flow through the column length will differ depending on the component's respective affinities, resulting in separation of the sample into discrete components. After exiting the column, the chemicals pass through a detector, such as a flame photometer or mass spectrometer, generating a signal proportional to the concentration. Since the retention time (rt) is characteristic of a specific compound, the rt can be use to identify components of the mixture by comparing with known rts, eliminating false alarms from similar compounds that have different rts. A preconcentrator specific to the analyte can also reduce false alarms caused by interferents. The preconcentrator passes air through an absorbent filter that traps agent molecules. The filter is then isolated from the air, connected to the GC, and heated to release any CA that may have been trapped. Two instruments that use gas chromatography are the Voyager Portable Gas Chromatograph from Photovac, Inc., (figure 15) and the CMS200, INFICON from (figure 16).

Figure 15 — Voyager Portable Ga

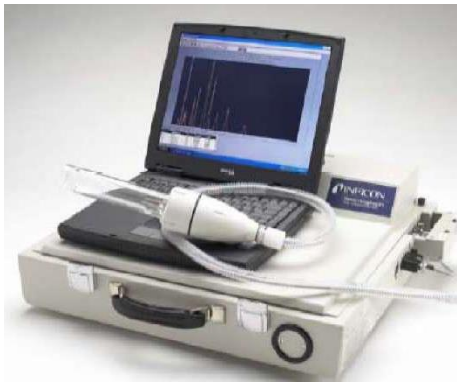

Figure 16 — CMS200, Chromatograph, Photovac, Inc.INFICON

Mass Spectrometry

Mass spectrometry (MS) is a technique that can positively identify a CA at very low concentrations. In this technique, a volatilized sample is introduced into a vacuum chamber and ionized by an electron beam. This electron impact ionization generates a molecular ion of the compound and also causes the molecule to split into a number of fragment ions characteristic of the sample. The ionized molecules and fragments are mass analyzed by rapidly scanning a quadrupole mass filter across a wide mass range, resulting in a spectrum of intensity versus ion mass-to-charge ratio (equivalent to mass for the singly charged ions usually observed). The identity of the substances can then be determined by comparing the mass spectrum with library spectra and computer searching or by detailed interpretation of the ion masses and ratios. Since each molecule forms a unique set of fragments, mass spectroscopy provides positive and unambiguous identification of pure compounds. However, mixed samples may be problematic and complicate spectral interpretation. To simplify interpretation of the mass spectrum, it is often necessary to separate the components in the sample, such as in GC/MS, in which the gas chromatograph column exit is connected directly to the inlet of the mass spectrometer to permit MS analysis of mixtures separated by the GC. Two instruments that use mass spectrometry are the Hapsite® manufactured by INFICON and the Agilent 6890N-5975B GC/MSD from Agilent Technologies (figure 17 and figure 18, respectively).

Figure 17 — Hapsite®, INFICON

Figure 18 — Agilent 6890N-5975BGC/MSD, Agilent Technologies

High-Performance Liquid Chromatography

High-performance liquid chromatography (HPLC) is most useful in the detection and identification of larger molecular weight CAs, or chemicals such as 3-quinuclidinyl benzilate [(QNB) BZ] or as lysergic acid diethylamide (LSD), and in the detection and identification of biological agents. With HPLC, compounds that do not easily volatilize can be analyzed without undergoing chemical derivatization. A solution of the sample is passed through a narrow bore column at high pressure, and species are separated based on their differential affini-

ty for the stationary phase packing in the column. The time spent (retention time) for each component of a mixture to flow through the column length will differ depending on the component's respective affinities, resulting in separation of the sample into discrete components. As with GCs, HPLC instruments can be equipped with a variety of detectors such as ultraviolet-visible (UV-VIS) spectrometers, mass spectrometers, fluorescence spectrometers, and electrochemical detectors. Limitations to the fielding of HPLCs and their detectors include the need for a 120 V ac source, the need for high purity solvents, and the size of the instruments. Currently there is no portable HPLC unit available.

Ion Chromatography

A chromatographic technique closely related to HPLC is ion chromatography (IC). In this technique, ionic species can be separated, detected, and identified. Limitations to the fielding of ICs and their detectors are similar to the limitations associated with fielding HPLC instrumentation, that is, IC instruments require power requirements (120 V ac source), high purity water, and high purity chemical reagents for the preparation of buffering solutions. Like HPLC, IC instruments can use UV-VIS spectrometers, mass spectrometers, and electrochemical detectors. Ion chromatography has been successfully used in the U.S. Army Materiel Command's Treaty Verification Laboratory in the analysis of several chemical nerve agents and their degradation products.

Capillary Zone Electrophoresis

Capillary zone electrophoresis (CZE or CE) is a chromatographic technique that can be thought of as a hybridization of gas chromatography, liquid chromatography, and ion chromatography. Rather than using a temperature gradient or a solvent gradient (as in GC or HPLC, respectively), a mobile phase containing an ionic buffer is used (as in ion chromatography). A high voltage electric field (either fixed potential or a gradient) is applied across a fused silica column similar to capillary columns used in GC.

The CZE instruments are typically configured with either a UV-VIS spectrometer or an electrochemical detector, but they can be interfaced to a mass spectrometer. The CZE instrumentation shares the same electrical requirements as HPLC and IC instruments. High purity water and chemical reagents are required but in much smaller quantities.

Ultraviolet Spectroscopy

Ultraviolet (UV) spectroscopy involves passing a monochromatic light through a dilute solution of the sample in a nonabsorbing solvent. The UV (UV = 200 nm to 400 nm) spectrum is generally taken by placing a dilute solu-

tion of the analyte in a silica cell and preparing a matching cell of pure solvent. The cells are placed in the spectrometer, and each cell is scanned with UV radiation. Ultraviolet spectra usually show only one broad peak indicating absorption. The intensity of the absorption is measured by the percent of the incident light that passes through the sample. The spectrum is determined by comparing the intensities of the transmitted light of the sample and the pure solvent.

CHAPTER 4
NERVE AGENTS

Among lethal CW agents, the nerve agents have had an entirely dominant role since the Second World War. Nerve agents acquired their name because they affect the transmission of nerve impulses in the nervous system. All nerve agents belong chemically to the group of organo-phosphorus compounds. They are stable and easily dispersed, highly toxic and have rapid effects both when absorbed through the skin and via respiration. Nerve agents can be manufactured by means of fairly simple chemical techniques. The raw materials are inexpensive and generally readily available.

It was not until the early 1930's that German chemists observed that organo-phosphorus compounds could be poisonous. In 1934, Dr Gerhard Schrader, a chemist at IG Farben, was given the task of developing a pesticide. Two years later a phosphorus compound with extremely high toxicity was produced for the first time. According to contemporary regulations, discoveries with implications had to be reported to the concerned authorities, which was also done with Schrader's discovery. This phosphorus compound, given the name tabun, was the first of the substances later referred to as nerve agents.

A factory for production of the new CW agent was built and a total of 12 000 tonnes of tabun were produced during the years 1942–1945. At the end of the war the Allies seized large quantities of this nerve agent. Up to the end of the war, Schrader and his co-workers synthesized about 2 000 new organo-phosphorus compounds, including sarin (1938). The third of the «classic» nerve agents, soman, was first produced in 1944. These three nerve agents are known as G agents in the American nomenclature. The manufacture of sarin never started properly and up to 1945 only about 0.5 tonne of this nerve agent was produced in a pilot plant.

Immediately after the war, research was mainly concentrated on studies of the mechanisms of the nerve agents in order to discover more effective forms of protection against these new CW agents. The results of these efforts led, however, not only to better forms of protection but also to new types of agents closely related to the earlier ones.

By the mid-1950's a group of more stable nerve agents had been developed, known as the V-agents in the American nomenclature. They are approximately

ten-fold more poisonous than sarin and are thus among the most toxic substances ever synthesized.

The first publication of these substances appeared in 1955. The authors, R. Ghosh and J. F. Newman, described one of the substances, known as Amiton, as being particularly effective against mites. At this time, intensive research was being devoted to the organo-phosphorus insecticides both in Europe and in the United States. At least three chemical firms appear to have independently discovered the remarkable toxicity of these phosphorus compounds during the years 1952–1953. Surprisingly enough, some of these substances were available on the market as pesticides. Nonetheless, they were soon withdrawn owing to their considerable toxicity also to mammals.

In the United States, the choice fell in 1958 on a substance known by its code name VX as suitable as a CW agent of persistent type. Full-scale production of VX started in April 1961 but its structure was not published until 1972.

SARIN (GB)

Common Names:

Trilone

Agent Characteristics

APPEARANCE: Clear, colorless liquid.

DESCRIPTION: Sarin (military designation GB) is a nerve agent that is one of the most toxic of the known chemical warfare agents. It is generally odorless and tasteless. Exposure to sarin can cause death in minutes. A fraction of an ounce (1 to 10 mL) of sarin on the skin can be fatal. Nerve agents are chemically similar to organophosphate pesticides and exert their effects by interfering with the normal function of the nervous system.

METHODS OF DISSEMINATION:

Indoor Air: Sarin can be released into indoor air as a liquid spray (aerosol) or as a vapor.

Water: Sarin can contaminate water.

Food: Sarin can contaminate food.

Outdoor Air: Sarin can be released into outdoor air as a liquid spray (aerosol) or as a vapor.

Agricultural: If sarin is released into the air as a liquid spray (aerosol), it has the potential to contaminate agricultural products. If sarin is released as a vapor, it is highly unlikely to contaminate agricultural products.

ROUTES OF EXPOSURE: Sarin can be absorbed into the body by inhalation, ingestion, skin contact, or eye contact. Ingestion is an uncommon route of exposure.

Personal Protective Equipment

GENERAL INFORMATION: First Responders should use a NIOSH-certified Chemical, Biological, Radiological, Nuclear (CBRN) Self Contained Breathing Apparatus (SCBA) with a Level A protective suit when entering an area with an unknown contaminant or when entering an area where the concentra-

tion of the contaminant is unknown. Level A protection should be used until monitoring results confirm the contaminant and the concentration of the contaminant.

NOTE: Safe use of protective clothing and equipment requires specific skills developed through training and experience.

LEVEL A: (RED ZONE): Select when the greatest level of skin, respiratory, and eye protection is required. This is the maximum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than the AEGL-2.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A Totally-Encapsulating Chemical Protective (TECP) suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, and a hard hat worn under the TECP suit are optional items.

LEVEL B: (RED ZONE): Select when the highest level of respiratory protection is necessary but a lesser level of skin protection is required. This is the minimum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than AEGL-2. It differs from Level A in that it incorporates a non-encapsulating, splash-protective, chemical-resistant splash suit that provides Level A protection against liquids but is not airtight.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL C: (YELLOW ZONE): Select when the contaminant and concentration of the contaminant are known and the respiratory protection criteria factors for using Air Purifying Respirators (APR) or Powered Air Purifying Respirators (PAPR) are met. This level is appropriate when decontaminating patient/victims.

A NIOSH-certified CBRN tight-fitting APR with a canister-type gas mask or CBRN PAPR for air levels greater than AEGL-2.

A NIOSH-certified CBRN PAPR with a loose-fitting face-piece, hood, or helmet and a filter or a combination organic vapor, acid gas, and particulate car-

tridge/filter combination or a continuous flow respirator for air levels greater than AEGL-1.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Escape mask, face shield, coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL D: (GREEN ZONE): Select when the contaminant and concentration of the contaminant are known and the concentration is below the appropriate occupational exposure limit or less than AEGL-1 for the stated duration times.

Limited to coveralls or other work clothes, boots, and gloves.

Emergency Response

CHEMICAL DANGERS:

Under acid conditions, sarin hydrolyzes to form hydrofluoric acid (HF). See the emergency response card for hydrofluoric acid.

Sarin decomposes tin, magnesium, cadmium-plated steel, and aluminum.

Contact with metals may evolve flammable hydrogen gas.

EXPLOSION HAZARDS:

When heated, vapors may form explosive mixtures with air, presenting an explosion hazard indoors, outdoors, and in sewers.

Containers may explode when heated.

FIRE FIGHTING INFORMATION:

Sarin is combustible.

The agent may burn but does not ignite readily.

Fire may produce irritating, corrosive, and/or toxic gases.

For small fires, use dry chemical, carbon dioxide, or water spray.

For large fires, use dry chemical, carbon dioxide, alcohol-resistant foam, or water spray. Move containers from the fire area if it is possible to do so without risk to personnel. Dike fire control water for later disposal; do not scatter the material.

Avoid methods that will cause splashing or spreading.

For fire involving tanks or car/trailer loads, fight the fire from maximum distance or use unmanned hose holders or monitor nozzles. Do not get water inside containers. Cool containers with flooding quantities of water until well after the fire is out. Withdraw immediately in case of rising sound from venting safety devices or discoloration of tanks. Always stay away from tanks engulfed in fire.

Run-off from fire control or dilution water may be corrosive and/or toxic, and it may cause pollution.

If the situation allows, control and properly dispose of run-off (effluent).

INITIAL ISOLATION AND PROTECTIVE ACTION DISTANCES:

If a tank, rail car, or tank truck is involved in a fire, isolate it for 0.5 mi (800 m) in all directions; also, consider initial evacuation for 0.5 mi (800 m) in all directions.

Small spills (involving the release of approximately 52.83 gallons (200 liters) or less), when sarin (GB) is used as a weapon

First isolate in all directions: 500 ft (150 m).

Then protect persons downwind during the day: 1.0 mi (1.7 km).

Then protect persons downwind during the night: 2.1 mi (3.4 km).

Large spills (involving quantities greater than 52.83 gallons (200 liters)), when sarin (GB) is used as a weapon

First isolate in all directions: 3000 ft (1000 m).

Then protect persons downwind during the day: 7.0+ mi (11.0+ km).

Then protect persons downwind during the night: 7.0+ mi (11.0+ km). ("+" means distance can be larger in certain atmospheric conditions.)

PHYSICAL DANGERS:

Vapors are heavier than air. They will spread along the ground and collect and stay in poorly-ventilated, low-lying, or confined areas (e.g., sewers, basements, and tanks).

Hazardous concentrations may develop quickly in enclosed, poorly-ventilated, or low-lying areas. Keep out of these areas. Stay upwind.

NFPA 704 Signal:

Health: 4

Flammability: 1

Reactivity: 0

Special:

Signs/Symptoms

TIME COURSE: Exposure to nerve agents may be rapidly fatal. Eye exposure: Liquid sarin produces health effects within seconds to minutes; larger exposures may cause death within 1 to 10 minutes. Ingestion exposure: No information is available on the time course of effects following ingestion of sarin. Inhalation exposure: Inhaled sarin produces health effects within seconds to minutes; larger exposures may cause death within 1 to 10 minutes. Skin exposure: Liquid sarin may produce health effects within minutes. Health effects from mild to moderate exposure may be delayed up to 18 hours; larger exposures may cause death within minutes to hours.

EFFECTS OF SHORT-TERM (LESS THAN 8-HOURS) EXPOSURE: Nerve agents cause the same health effects regardless of the route of exposure. Initial effects depend on the dose and route of exposure. Nerve agents interfere with the normal functioning of the nervous system. Skeletal muscles, certain organs of the body, and the central nervous system (CNS) may all be affected by exposure to the nerve agent.

EYE EXPOSURE:

Contracted or pinpoint pupils (miosis), redness of the membranes (conjunctiva), pain in and around the eye, dim and/or blurred vision, sensation of pressure with heaviness, and reflex nausea and vomiting (emesis).

Effects are usually local, occuring from direct contact with nerve agent vapor, aerosol, or liquid; but exposure by other routes can also affect the eyes.

INGESTION EXPOSURE:

Nausea, vomiting (emesis), diarrhea, abdominal pain, and cramping.

INHALATION EXPOSURE:

Mild to moderate: Contracted or pinpoint pupils (miosis), runny nose (rhinorrhea), narrowing of the large airways (bronchoconstriction), fluid accumulation within the airways of the lungs, and slight to moderate difficulty breathing or shortness of breath (dyspnea).

Severe: In addition to the symptoms described above, there can be loss of consciousness; seizures; muscular twitching (fasciculations); floppy (flaccid) paralysis; increased fluid accumulation within the airways and within the digestive tract, resulting in secretions from the nose and mouth; cessation of breathing (apnea); and death.

SKIN EXPOSURE:

Mild to moderate: Health effects may be immediate or may be delayed up to 18 hours. Profuse sweating (diaphoresis) and muscular twitching (fasciculations) at the site of contact, nausea, vomiting (emesis), diarrhea, and weakness (malaise).

Severe: Health effects may appear quickly; 2 to 30 minutes post-exposure. In addition to the above, there can be loss of consciousness, seizures, muscular twitching (fasciculations), floppy (flaccid) paralysis, increased fluid accumulation within the airways and within the digestive tract resulting in secretions from the nose and mouth, cessation of breathing (apnea), and death.

Decontamination

INTRODUCTION: The purpose of decontamination is to make an individual and/or their equipment safe by physically removing toxic substances quickly and effectively. Care should be taken during decontamination, because absorbed agent can be released from clothing and skin as a gas. Your Incident Commander will provide you with decontaminants specific for the agent released or the agent believed to have been released.

DECONTAMINATION CORRIDOR: The following are recommendations to protect the first responders from the release area:

Position the decontamination corridor upwind and uphill of the hot zone.

The warm zone should include two decontamination corridors. One decontamination corridor is used to enter the warm zone and the other for exiting the warm zone into the cold zone. The decontamination zone for exiting should be upwind and uphill from the zone used to enter.

Decontamination area workers should wear appropriate PPE. See the PPE section of this card for detailed information.

A solution of detergent and water (which should have a pH value of at least 8 but should not exceed a pH value of 10.5) should be available for use in decontamination procedures. Soft brushes should be available to remove contamination from the PPE.

Labeled, durable 6-mil polyethylene bags should be available for disposal of contaminated PPE.

INDIVIDUAL DECONTAMINATION: The following methods can be used to decontaminate an individual:

Decontamination of First Responder:

Begin washing PPE of the first responder using soap and water solution and a soft brush. Always move in a downward motion (from head to toe). Make sure to get into all areas, especially folds in the clothing. Wash and rinse (using cold or warm water) until the contaminant is thoroughly removed.

Remove PPE by rolling downward (from head to toe) and avoid pulling PPE off over the head. Remove the SCBA after other PPE has been removed.

Place all PPE in labeled durable 6-mil polyethylene bags.

Decontamination of Patient/Victim:

Remove the patient/victim from the contaminated area and into the decontamination corridor.

Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag.

Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds.

Cover the patient/victim to prevent shock and loss of body heat.

Move the patient/victim to an area where emergency medical treatment can be provided.

First Aid

GENERAL INFORMATION: Administration of antidotes is a critical step in managing a patient/victim. However, this may be difficult to achieve in the Red Zone, because the antidotes may not be readily available, and procedures or policies for their administration in the Red Zone may be lacking. Do not administer antidotes preventatively; there is no benefit to doing so. Diazepam (or other benzodiazepines) should be administered when there is evidence of seizures, usually seen in cases of moderate to severe exposure to a nerve agent. Remember, physical findings of localized exposure often precede systemic exposure and physical findings.

ANTIDOTE: Atropine and pralidoxime chloride (2-PAM Cl) are antidotes for nerve agent toxicity; however, 2-PAM Cl must be administered within minutes to a few hours (depending on the agent) following exposure to be effective. There is also generally no benefit in giving more than three injections of 2-

PAM Cl. Atropine should be administered every 5 to 10 minutes until secretions begin to dry up. If the military Mark I kits containing autoinjectors are available, they provide the best way to administer the antidotes to healthy adults. One autoinjector automatically delivers 2 mg atropine and the other automatically delivers 600 mg 2-PAM Cl. If the Mark I kit is unavailable, or the patient/victim is not an otherwise healthy adult, administer antidotes as described below:

Infant (0–2 yrs), for mild to moderate physical findings, including localized sweating, muscular twitching (fasciculations), nausea, vomiting, weakness, and shortness of breath (dyspnea); administer Atropine at 0.05 mg/kg IM; 2-PAM Cl at 15 mg/kg IM.

Infant (0–2 yrs), for severe physical findings, including unconsciousness, convulsions, cessation of breathing (apnea), and floppy (flaccid) paralysis; administer Atropine at 0.1 mg/kg IM; 2-PAM Cl at 25 mg/kg IM.

Child (2–10 yrs), for mild to moderate physical findings, including localized sweating, muscular twitching (fasciculations), nausea, vomiting, weakness, and shortness of breath (dyspnea); administer Atropine at 1 mg/kg IM; 2-PAM Cl at 15 mg/kg IM.

Child (2–10 yrs), for severe physical findings, including unconsciousness, convulsions, cessation of breathing (apnea), and floppy (flaccid) paralysis; administer Atropine at 2 mg/kg IM; 2-PAM Cl at 25 mg/kg IM.

Adolescent (> 10 yrs), for mild to moderate physical findings, including localized sweating, muscular twitching (fasciculations), nausea, vomiting, weakness, and shortness of breath (dyspnea); administer Atropine at 2 mg/kg IM; 2-PAM Cl at 15 mg/kg IM.

Adolescent (> 10 yrs), for severe physical findings, including unconsciousness, convulsions, cessation of breathing (apnea), and floppy (flaccid) paralysis; administer Atropine at 4 mg IM; 2-PAM Cl at 25 mg/kg IM.

Adult, for mild to moderate physical findings, including localized sweating, muscular twitching (fasciculations), nausea, vomiting, weakness, and shortness of breath (dyspnea); administer Atropine at 2 to 4 mg IM; 2-PAM Cl at 600 mg IM.

Adult, for severe physical findings, including unconsciousness, convulsions, cessation of breathing (apnea), and floppy (flaccid) paralysis; administer Atropine at 6 mg IM; 2-PAM Cl at 1800 mg IM.

Elderly, frail for mild to moderate physical findings, including localized sweating, muscular twitching (fasciculations), nausea, vomiting, weakness, and shortness of breath (dyspnea); administer Atropine at 1 mg IM; 2-PAM Cl at 10 mg/kg IM.

Elderly, frail for severe physical findings, including unconsciousness, convulsions, cessation of breathing (apnea), and floppy (flaccid) paralysis; administer Atropine at 2 to 4 mg IM; 2-PAM Cl at 25 mg/kg IM.

Assisted ventilation should be started after administration of antidotes for severe exposures.

Repeat atropine (2 mg IM for adults or 0.05 to 0.1 mg/kg for children) at 5 to 10 minute intervals until secretions have diminished and breathing is comfortable or airway resistance has returned to near normal.

EYE:

Immediately remove the patient/victim from the source of exposure.

Often the first physical finding of minimal symptomatic exposure to nerve agent vapor is markedly constricted pupils (miosis); however, if this is the only physical finding of nerve agent exposure, do not administer antidotes but follow the instructions below.

When exposed to liquid nerve agent, immediately flush the eyes with water for about 5 to 10 minutes by tilting the head to the side, pulling the eyelids apart with fingers, and pouring water slowly into the eyes.

When exposed to nerve agent vapor, there is no need to flush the eyes.

Do not cover eyes with bandages.

Changes in the eye can lead to nausea and vomiting without necessarily being a sign of systemic exposure. However, if eye pain, nausea, or vomiting are seen in combination with any other physical findings of nerve agent poisoning, administer antidotes atropine and 2-PAM Cl as directed.

Seek medical attention immediately.

INGESTION:

Immediately remove the patient/victim from the source of exposure.

Ensure that the patient/victim has an unobstructed airway.

Do not induce vomiting (emesis).

Administer nothing by mouth (NPO).

If the patient/victim's condition can be evaluated within 30 minutes of ingestion, in a hospital setting, consider gastric lavage. Gastric contents should be considered potentially hazardous and should be quickly isolated.

Be alert to physical findings of systemic exposure, and administer antidotes as required.

Maintain records of all injections given.

Seek medical attention immediately.

INHALATION:

Immediately remove the patient/victim from the source of exposure.

In cases of moderate to severe exposure, antidotes alone will not provide effective treatment, and ventilatory support is essential.

Evaluate respiratory function and pulse.

Ensure that the patient/victim has an unobstructed airway.

Assist with ventilation as required. Do not provide mouth-to-mouth resuscitation. Contact with off-gassed vapor or with liquid agent may occur.

If shortness of breath occurs, or breathing is difficult (dyspnea), administer oxygen.

Suction secretions from the nose, mouth, and respiratory tract.

Marked resistance to ventilation is expected due to bronchial constriction and spasm. Resistance lessens after administration of atropine.

Ventilatory distress is a physical finding of systemic exposure and requires antidote administration.

Maintain records of all injections given.

Seek medical attention immediately.

SKIN:

Immediately remove the patient/victim from the source of exposure.

Some nerve agents may remain in the hair or clothing and should be decontaminated, if that was not previously done. See the decontamination section of this card.

Skin exposure to liquid nerve agents will not necessarily result in systemic exposure if the site of exposure is decontaminated promptly. Before administering nerve agent antidotes, observe the site of exposure for localized sweating and muscular twitching. If these physical findings appear, administer antidotes; otherwise careful observation is all that is needed.

Maintain records of all injections given.

Seek medical attention immediately.

See ATSDR Medical Management Guidelines for Nerve Agents (https://www.atsdr.cdc.gov/MHMI/mmg166.pdf)for more detailed recommendations.

Long-Term Implications

MEDICAL TREATMENT: Electrocardiogram (ECG), and adequacy of respiration and ventilation, should be monitored. Supplemental oxygenation, frequent suctioning of secretions, insertion of a tube into the trachea (endotracheal intubation), and assisted ventilation may be required. Diazepam (5 to 10 mg in adults and 0.2 to 0.5 mg/kg in children) may be used to control convulsions. Lorazepam or other benzodiazepines may be used, but barbiturates, phenytoin, and other anticonvulsants are not effective. Administration of atropine (if not already given) should precede the administration of benzodiazepines in order to best control seizures. Patients/victims who have inhalation exposure and who complain of chest pain, chest tightness, or cough should be observed and examined periodically for 6 to 12 hours to detect delayed-onset inflammation of the large airways (bronchitis), inflammatory lung disease (pneumonia), accumulation of fluid in the lungs (pulmonary edema), or respiratory failure.

DELAYED EFFECTS OF EXPOSURE: Patients/victims who have severe exposure should be evaluated for persistent central nervous system (CNS) effects.

EFFECTS OF CHRONIC OR REPEATED EXPOSURE: Limited data are available on chronic or repeated exposure to sarin. The available data however, suggest that sarin is not a human carcinogen, reproductive toxin, or developmental toxin. Limited data suggest that chronic or repeated exposure to sarin may result in a delayed postural sway and/or impaired psychomotor performance (neuropathy).

On-Site Fatalities

INCIDENT SITE:

Consult with the Incident Commander regarding the agent dispersed, dissemination method, level of PPE required, location, geographic complications (if any), and the approximate number of remains.

Coordinate responsibilities and prepare to enter the scene as part of the evaluation team along with the FBI HazMat Technician, local law enforcement evidence technician, and other relevant personnel.

Begin tracking remains using waterproof tags.

RECOVERY AND ON-SITE MORGUE:

Wear PPE until all remains are deemed free of contamination.

Establish a preliminary (holding) morgue.

Gather evidence, and place it in a clearly labeled impervious container. Hand any evidence over to the FBI.

Remove and tag personal effects.

Perform a thorough external evaluation and a preliminary identification check.

See the Decontamination section for decontamination procedures.

Decontaminate remains before they are removed from the incident site.

ENVIRONMENT/SPILLAGE DISPOSAL: The following methods can be used to decontaminate the environment/spillage disposal:

Do not touch or walk through the spilled agent if at all possible. However, if you must, personnel should wear the appropriate PPE during environmental decontamination. See the PPE section of this card for detailed information.

Keep combustibles (e.g., wood, paper, and oil) away from the spilled agent.

Use water spray to reduce vapors or divert vapor cloud drift. Avoid allowing water runoff to contact the spilled agent.

Do not direct water at the spill or the source of the leak.

Stop the leak if it is possible to do so without risk to personnel, and turn leaking containers so that gas rather than liquid escapes.

Prevent entry into waterways, sewers, basements, or confined areas.

Isolate the area until gas has dispersed.

Ventilate the area.

EQUIPMENT: Agents can seep into the crevices of equipment making it dangerous to handle. The following methods can be used to decontaminate equipment:

Not established/determined

Agent Properties

Chemical Formula:

$C_4H_{10}FO_2P$

Aqueous solubility:

Soluble

Boiling Point:

297 °F (147 °C)

Density:

Liquid: 1.10 g/mL at 68 °F (20 °C)

Vapor: 4.9 (air = 1)

Flammability:

Combustible

Flashpoint:
>536 °F (>280 °C)
172.4 °F (78 °C) (closed cup method)
Ionization potential:
Not established/determined
Log $K_{benzene\text{-}water}$:
Not established/determined
Log K_{ow} (estimated):
-1.4
Melting Point:
-70.6 °F (-57 °C)
Molecular Mass:
140.09
Soluble In:
All solvents
Specific Gravity:
1.089
Vapor Pressure:
2.1 mm Hg at 68 °F (20 °C)
2.9 mm Hg at 77 °F (25 °C)
Volatility:
22,000 mg/m^3 at 77 °F (25 °C)

SOMAN (GD)
Common Names:
Pinacolyl methylfluorophosphonate
Agent Characteristics

APPEARANCE: Clear, colorless, liquid. Discolors with aging to dark brown. Gives off colorless vapor.

DESCRIPTION: Soman (military designation GD) is one of the nerve agents, which are the most toxic of the known chemical warfare agents. It has an odor like camphor or rotting fruit. Exposure to soman can cause death in minutes. A fraction of an ounce (1 to 10 mL) of soman on the skin can be fatal. Nerve agents are chemically similar to organophosphate pesticides and exert their effects by interfering with the normal function of the nervous system.

METHODS OF DISSEMINATION:

Indoor Air: Soman can be released into indoor air as a liquid spray (aerosol) or as a vapor.

Water: Soman can contaminate water, although it will decompose slowly.

Food: Soman can contaminate food.

Outdoor Air: Soman can be released into outdoor air as a liquid spray (aerosol) or as a vapor.

Agricultural: If soman is released into the air as a liquid spray (aerosol), it has the potential to contaminate agricultural products. If soman is released as a vapor, it is highly unlikely to contaminate agricultural products.

ROUTES OF EXPOSURE: Soman can be absorbed into the body by inhalation, ingestion, skin contact, or eye contact. Ingestion is an uncommon route of exposure.

Personal Protective Equipment.

GENERAL INFORMATION: First Responders should use a NIOSH-certified Chemical, Biological, Radiological, Nuclear (CBRN) Self Contained Breathing Apparatus (SCBA) with a Level A protective suit when entering an area with an unknown contaminant or when entering an area where the concentration of the contaminant is unknown. Level A protection should be used until monitoring results confirm the contaminant and the concentration of the contaminant.

NOTE: Safe use of protective clothing and equipment requires specific skills developed through training and experience.

LEVEL A: (RED ZONE): Select when the greatest level of skin, respiratory, and eye protection is required. This is the maximum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than the AEGL-2.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A Totally-Encapsulating Chemical Protective (TECP) suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, and a hard hat worn under the TECP suit are optional items.

LEVEL B: (RED ZONE): Select when the highest level of respiratory protection is necessary but a lesser level of skin protection is required. This is the minimum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than AEGL-2. It differs from Level A in that it incorporates a non-encapsulating, splash-protective, chemical-resistant splash suit that provides Level A protection against liquids but is not airtight.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL C: (YELLOW ZONE): Select when the contaminant and concentration of the contaminant are known and the respiratory protection criteria factors for using Air Purifying Respirators (APR) or Powered Air Purifying Respirators (PAPR) are met. This level is appropriate when decontaminating patient/victims.

A NIOSH-certified CBRN tight-fitting APR with a canister-type gas mask or CBRN PAPR for air levels greater than AEGL-2.

A NIOSH-certified CBRN PAPR with a loose-fitting face-piece, hood, or helmet and a filter or a combination organic vapor, acid gas, and particulate cartridge/filter combination or a continuous flow respirator for air levels greater than AEGL-1.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Escape mask, face shield, coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL D: (GREEN ZONE): Select when the contaminant and concentration of the contaminant are known and the concentration is below the appropriate occupational exposure limit or less than AEGL-1 for the stated duration times.

Limited to coveralls or other work clothes, boots, and gloves.

Emergency Response

CHEMICAL DANGERS:

Under acid conditions, soman hydrolyzes to form hydrofluoric acid (HF). See the emergency response card for hydrofluoric acid.

Soman is destroyed by bleaching powder, but the reaction produces cyanogen chloride (CNCl). See the emergency response card for cyanogen chloride.

Soman reacts readily with bases and weak acids.

When heated to decomposition, soman can emit highly toxic fumes.

Soman decomposes slowly in water. Raising the pH increases the rate of decomposition significantly.

Contact with metals may evolve flammable hydrogen gas.

EXPLOSION HAZARDS:

When heated, vapors may form explosive mixtures with air, presenting an explosion hazard indoors, outdoors, and in sewers.

Containers may explode when heated.

FIRE FIGHTING INFORMATION:

Soman is combustible.

The agent may burn but does not ignite readily.

Fire may produce irritating, corrosive, and/or toxic gases.

For small fires, use dry chemical, carbon dioxide, or water spray.

For large fires, use dry chemical, carbon dioxide, alcohol-resistant foam, or water spray. Move containers from the fire area if it is possible to do so without risk to personnel. Dike fire control water for later disposal; do not scatter the material.

Avoid methods that will cause splashing or spreading.

For fire involving tanks or car/trailer loads, fight the fire from maximum distance or use unmanned hose holders or monitor nozzles. Do not get water inside containers. Cool containers with flooding quantities of water until well after the fire is out. Withdraw immediately in case of rising sound from venting safety devices or discoloration of tanks. Always stay away from tanks engulfed in fire.

Runoff from fire control or dilution water may be corrosive and/or toxic, and it may cause pollution.

If the situation allows, control and properly dispose of run-off (effluent).

INITIAL ISOLATION AND PROTECTIVE ACTION DISTANCES:

If a tank, rail car, or tank truck is involved in a fire, isolate it for 0.5 mi (800 m) in all directions; also, consider initial evacuation for 0.5 mi (800 m) in all directions.

Small spills (involving the release of approximately 52.83 gallons (200 liters) or less), when soman (thickened GD) is used as a weapon

First isolate in all directions: 300 ft (90 m).

Then protect persons downwind during the day: 0.5 mi (0.9 km).

Then protect persons downwind during the night: 1.1 mi (1.8 km).

Large spills (involving quantities greater than 52.83 gallons (200 liters)), when soman (thickened GD) used as a weapon

First isolate in all directions: 2500 ft (800 m).

Then protect persons downwind during the day: 4.2 mi (6.8 km).

Then protect persons downwind during the night: 6.5 mi (10.5 km).

PHYSICAL DANGERS:

Vapors are heavier than air. They will spread along the ground and collect and stay in poorly-ventilated, low-lying, or confined areas (e.g., sewers, basements, and tanks).

Hazardous concentrations may develop quickly in enclosed, poorly-ventilated, or low-lying areas. Keep out of these areas. Stay upwind.

NFPA 704 Signal:

Health: 4

Flammability: 1

Reactivity: 0

Special:

Signs/Symptoms

TIME COURSE: Exposure to nerve agents may be rapidly fatal. Eye exposure: Liquid soman produces health effects within seconds to minutes; larger exposures may cause death within 1 to 10 minutes. Ingestion exposure: No information is available on the time course of effects following ingestion of soman. Inhalation exposure: Inhaled soman produces health effects within seconds to minutes; larger exposures may cause death within 1 to 10 minutes. Skin exposure: Liquid soman may produce health effects within minutes. Health effects from mild to moderate exposure may be delayed up to 18 hours; larger exposures may cause death within minutes to hours.

EFFECTS OF SHORT-TERM (LESS THAN 8-HOURS) EXPOSURE: Nerve agents cause the same health effects regardless of the route of exposure. Initial effects depend on the dose and route of exposure. Nerve agents interfere with the normal functioning of the nervous system. Skeletal muscles, certain organs of the body, and the central nervous system (CNS) may all be affected by exposure to nerve agent.

EYE EXPOSURE:

Contracted or pinpoint pupils (miosis), redness of the membranes (conjunctiva), pain in and around the eye, dim and/or blurred vision, sensation of pressure with heaviness, reflex nausea and vomiting (emesis).

Effects are usually local, occuring from direct contact with nerve agent vapor, aerosol, or liquid, but exposure by other routes can also affect the eyes.

INGESTION EXPOSURE:

Nausea, vomiting (emesis), diarrhea, abdominal pain, cramping.

INHALATION EXPOSURE:

Mild to moderate: Contracted or pinpoint pupils (miosis), runny nose (rhinorrhea), narrowing of the large airways (bronchoconstriction), fluid accumulation within the airways of the lungs, and slight to moderate difficulty breathing or shortness of breath (dyspnea).

Severe: In addition to the symptoms described above, there can be loss of consciousness, seizures, muscular twitching (fasciculations), floppy (flaccid) paralysis, increased fluid accumulation within the airways and within the digestive tract, resulting in secretions from the nose and mouth, cessation of breathing (apnea), and death.

SKIN EXPOSURE:

Mild to moderate: Health effects may be immediate or may be delayed up to 18 hours. Profuse sweating (diaphoresis) and muscular twitching (fasciculations) at the site of contact, nausea, vomiting (emesis), diarrhea, and weakness (malaise).

Severe: Health effects may appear quickly; 2 to 30 minutes post-exposure. In addition to the symptoms described above, there can be loss of consciousness, seizures, muscular twitching (fasciculations), floppy (flaccid) paralysis, increased fluid accumulation within the airways and within the digestive tract resulting in secretions from the nose and mouth, cessation of breathing (apnea), and death.

Decontamination.

INTRODUCTION: The purpose of decontamination is to make an individual and/or their equipment safe by physically removing toxic substances quickly and effectively. Care should be taken during decontamination, because absorbed agent can be released from clothing and skin as a gas. Your Incident Commander will provide you with decontaminants specific for the agent released or the agent believed to have been released.

DECONTAMINATION CORRIDOR: The following are recommendations to protect the first responders from the release area:

Position the decontamination corridor upwind and uphill of the hot zone.

The warm zone should include two decontamination corridors. One decontamination corridor is used to enter the warm zone and the other for exiting the warm zone into the cold zone. The decontamination zone for exiting should be upwind and uphill from the zone used to enter.

Decontamination area workers should wear appropriate PPE. See the PPE section of this card for detailed information.

A solution of detergent and water (which should have a pH value of at least 8 but should not exceed a pH value of 10.5) should be available for use in decontamination procedures. Soft brushes should be available to remove contamination from the PPE.

Labeled, durable 6-mil polyethylene bags should be available for disposal of contaminated PPE.

INDIVIDUAL DECONTAMINATION: The following methods can be used to decontaminate an individual:

Decontamination of First Responder:

Begin washing PPE of the first responder using soap and water solution and a soft brush. Always move in a downward motion (from head to toe). Make sure to get into all areas, especially folds in the clothing. Wash and rinse (using cold or warm water) until the contaminant is thoroughly removed.

Remove PPE by rolling downward (from head to toe) and avoid pulling PPE off over the head. Remove the SCBA after other PPE has been removed.

Place all PPE in labeled durable 6-mil polyethylene bags.

Decontamination of Patient/Victim:

Remove the patient/victim from the contaminated area and into the decontamination corridor.

Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag.

Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds.

Cover the patient/victim to prevent shock and loss of body heat.

Move the patient/victim to an area where emergency medical treatment can be provided.

First Aid.

GENERAL INFORMATION: Initial treatment consists of repeated administration of antidotes and supportive measures.

ANTIDOTE: Atropine and pralidoxime chloride (2-PAM Cl) are antidotes for nerve agent toxicity; however, 2-PAM Cl must be administered within minutes to a few hours (depending on the agent) following exposure to be effective. There is also generally no benefit in giving more than three injections of 2-PAM Cl. Atropine should be administered every 5 to 10 minutes until secretions begin to dry up. If the military Mark I kits containing autoinjectors are available, they provide the best way to administer the antidotes to healthy adults. One autoinjector automatically delivers 2 mg atropine and the other automatically delivers 600 mg 2-PAM Cl. If the Mark I kit is unavailable, or the patient/victim is not an otherwise healthy adult, administer antidotes as described below:

Repeat atropine (2 mg IM for adults or 0.05 to 0.1 mg/kg for children) at 5 to 10 minute intervals until secretions have diminished and breathing is comfortable or airway resistance has returned to near normal.

EYE:

Immediately remove the patient/victim from the source of exposure.

Often the first physical finding of minimal symptomatic exposure to nerve agent vapor is markedly constricted pupils (miosis); however, if this is the only physical finding of nerve agent exposure, do not administer antidotes but follow the instructions below.

When exposed to liquid nerve agent, immediately flush the eyes with water for about 5 to 10 minutes by tilting the head to the side, pulling the eyelids apart with fingers, and pouring water slowly into eyes.

When exposed to nerve agent vapor, there is no need to flush the eyes.

Do not cover eyes with bandages.

Changes in the eye can lead to nausea and vomiting without necessarily being a sign of systemic exposure. However, if eye pain, nausea, or vomiting are seen in combination with any other physical findings of nerve agent poisoning, administer antidotes atropine and 2-PAM Cl as directed.

Seek medical attention immediately.

INGESTION:

Immediately remove the patient/victim from the source of exposure.

Ensure that the patient/victim has an unobstructed airway.

Do not induce vomiting (emesis).

Administer nothing by mouth (NPO).

If the patient/victim's condition can be evaluated within 30 minutes after ingestion, in a hospital setting, consider gastric lavage. Gastric contents should be considered potentially hazardous and should be quickly isolated.

Be alert to physical findings of systemic exposure, and administer antidotes as required.

Maintain records of all injections given.

Seek medical attention immediately.

INHALATION:

Immediately remove the patient/victim from the source of exposure.

In cases of moderate to severe exposure, antidotes alone will not provide effective treatment, and ventilatory support is essential.

Evaluate respiratory function and pulse.

Ensure that the patient/victim has an unobstructed airway.

Assist with ventilation as required. Do not provide mouth-to-mouth resuscitation. Contact with off-gassed vapor or with liquid agent may occur.

If shortness of breath occurs, or breathing is difficult (dyspnea), administer oxygen.

Suction secretions from the nose, mouth, and respiratory tract.

Marked resistance to ventilation is expected due to bronchial constriction and spasm. Resistance lessens after administration of atropine.

Ventilatory distress is a physical finding of systemic exposure and requires antidote administration.

Maintain records of all injections given.

Seek medical attention immediately.

SKIN:

Immediately remove the patient/victim from the source of exposure.

Some nerve agents may remain in the hair or clothing and should be decontaminated, if that was not previously done. See the decontamination section of this card.

Skin exposure to liquid nerve agents will not necessarily result in systemic exposure if the site of exposure is decontaminated promptly. Before administering nerve agent antidotes, observe the site of exposure for localized sweating and muscular twitching. If these physical findings appear, administer antidotes; otherwise careful observation is all that is needed.

Maintain records of all injections given.

Seek medical attention immediately.

See ATSDR Medical Management Guidelines for Nerve Agents for more detailed recommendations, https://www.atsdr.cdc.gov/MHMI/mmg166.pdf.

Long-Term Implications

MEDICAL TREATMENT: Electrocardiogram (ECG), and adequacy of respiration and ventilation, should be monitored. Supplemental oxygenation, frequent suctioning of secretions, insertion of a tube into the trachea (endotracheal intubation), and assisted ventilation may be required. Diazepam (5 to 10 mg in adults and 0.2 to 0.5 mg/kg in children) may be used to control convulsions. Lorazepam or other benzodiazepines may be used, but barbiturates, phenytoin, and other anticonvulsants are not effective. Administration of atropine (if not already given) should precede the administration of benzodiazepines in order to best control seizures. Patients/victims who have inhalation exposure and who complain of chest pain, chest tightness, or cough should be observed and examined periodically for 6 to 12 hours to detect delayed-onset inflammation of the large airways (bronchitis), inflammatory lung disease (pneumonia), accumulation of fluid in the lungs (pulmonary edema), or respiratory failure.

DELAYED EFFECTS OF EXPOSURE: Patients/victims who have severe exposure should be evaluated for persistent central nervous system (CNS) effects.

EFFECTS OF CHRONIC OR REPEATED EXPOSURE: Limited data are available on chronic or repeated exposure to soman. The available data however, suggest that soman is not a human carcinogen, reproductive toxin, or developmental toxin. Limited data suggest that chronic or repeated exposure to soman may result in a delayed postural sway and/or impaired psychomotor performance (neuropathy).

On-Site Fatalities.

INCIDENT SITE:

Consult with the Incident Commander regarding the agent dispersed, dissemination method, level of PPE required, location, geographic complications (if any), and the approximate number of remains.

Coordinate responsibilities and prepare to enter the scene as part of the evaluation team along with the FBI HazMat Technician, local law enforcement evidence technician, and other relevant personnel.

Begin tracking remains using waterproof tags.

RECOVERY AND ON-SITE MORGUE:

Wear PPE until all remains are deemed free of contamination.

Establish a preliminary (holding) morgue.

Gather evidence, and place it in a clearly labeled impervious container. Hand any evidence over to the FBI.

Remove and tag personal effects.

Perform a thorough external evaluation and a preliminary identification check.

See the Decontamination section for decontamination procedures.

Decontaminate remains before they are removed from the incident site.

ENVIRONMENT/SPILLAGE DISPOSAL: The following methods can be used to decontaminate the environment/spillage disposal:

Do not touch or walk through the spilled agent if at all possible. However, if you must, personnel should wear the appropriate PPE during environmental decontamination. See the PPE section of this card for detailed information.

Keep combustibles (e.g., wood, paper, and oil) away from the spilled agent.

Use water spray to reduce vapors or divert vapor cloud drift. Avoid allowing water runoff to contact the spilled agent.

Do not direct water at the spill or the source of the leak.

Stop the leak if it is possible to do so without risk to personnel, and turn leaking containers so that gas rather than liquid escapes.

Prevent entry into waterways, sewers, basements, or confined areas.

Isolate the area until gas has dispersed.

Ventilate the area.

EQUIPMENT: Agents can seep into the crevices of equipment making it dangerous to handle. The following methods can be used to decontaminate equipment:

Not established/determined
Agent Properties
Chemical Formula:
$C_7H_{16}FO_2P$
Aqueous solubility:
Slightly soluble
Boiling Point:
332.6 °F to 392 °F (167 °C to 200 °C)
Density:
Liquid: 1.02 g/mL at 77 °F (25 °C)
Vapor: 6.33 (air = 1)
Flammability:
Combustible
Flashpoint:
250 °F (121 °C)
Ionization potential:
Not established/determined
Log $K_{benzene\text{-}water}$:
Not established/determined
Log K_{ow} (estimated):
1.02
Melting Point:
-43.6 °F (-42 °C)
Molecular Mass:
182.17
Soluble In:
Lipids
Specific Gravity:
1.022
Vapor Pressure:
0.4 mm Hg at 77 °F (2 5°C)

VX
Common Names:
Methylphosphonothioic acid
O-ethyl S-(2-diisopropylaminoethyl) methylphosphonothiolate
Agent Characteristics
APPEARANCE: Clear, amber-colored, oily liquid.
DESCRIPTION: VX is one of the nerve agents, which are the most toxic of the known chemical warfare agents. It is tasteless and odorless. Exposure to VX can cause death in minutes. As little as one drop of VX on the skin can be fatal. Nerve agents are chemically similar to organophosphate pesticides and exert their effects by interfering with the normal function of the nervous system.

METHODS OF DISSEMINATION:

Indoor Air: VX can be released into indoor air as a liquid spray (aerosol) or as a vapor when temperatures are high.

Water: VX can contaminate water; it can break down in water to produce other toxic compounds.

Food: VX can contaminate food.

Outdoor Air: VX can be released into outdoor air as a liquid spray (aerosol) or as a vapor when temperatures are high.

Agricultural: If VX is released into the air as fine particles (aerosol), it has the potential to contaminate agricultural products. If VX is released as a vapor, it is highly unlikely to contaminate agricultural products.

ROUTES OF EXPOSURE: VX can be absorbed into the body by inhalation, ingestion, skin contact, or eye contact. Ingestion is an uncommon route of exposure.

Personal Protective Equipment

GENERAL INFORMATION: First Responders should use a NIOSH-certified Chemical, Biological, Radiological, Nuclear (CBRN) Self Contained Breathing Apparatus (SCBA) with a Level A protective suit when entering an area with an unknown contaminant or when entering an area where the concentration of the contaminant is unknown. Level A protection should be used until monitoring results confirm the contaminant and the concentration of the contaminant.

NOTE: Safe use of protective clothing and equipment requires specific skills developed through training and experience.

LEVEL A: (RED ZONE): Select when the greatest level of skin, respiratory, and eye protection is required. This is the maximum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than the AEGL-2.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A Totally-Encapsulating Chemical Protective (TECP) suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, and a hard hat worn under the TECP suit are optional items.

LEVEL B: (RED ZONE): Select when the highest level of respiratory protection is necessary but a lesser level of skin protection is required. This is the minimum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than AEGL-2. It differs from Level A in that it incorporates a non-encapsulating, splash-protective, chemical-resistant splash suit that provides Level A protection against liquids but is not airtight.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL C: (YELLOW ZONE): Select when the contaminant and concentration of the contaminant are known and the respiratory protection criteria factors for using Air Purifying Respirators (APR) or Powered Air Purifying Respirators (PAPR) are met. This level is appropriate when decontaminating patient/victims.

A NIOSH-certified CBRN tight-fitting APR with a canister-type gas mask or CBRN PAPR for air levels greater than AEGL-2.

A NIOSH-certified CBRN PAPR with a loose-fitting face-piece, hood, or helmet and a filter or a combination organic vapor, acid gas, and particulate cartridge/filter combination or a continuous flow respirator for air levels greater than AEGL-1.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Escape mask, face shield, coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL D: (GREEN ZONE): Select when the contaminant and concentration of the contaminant are known and the concentration is below the appropriate occupational exposure limit or less than AEGL-1 for the stated duration times.

Limited to coveralls or other work clothes, boots, and gloves.

Emergency Response

CHEMICAL DANGERS:

VX is on the Superfund Extremely Dangerous Substances list.

Contact with metals may evolve flammable hydrogen gas.

EXPLOSION HAZARDS:

When heated, vapors may form explosive mixtures with air, presenting an explosion hazard indoors, outdoors, and in sewers.

Containers may explode when heated.

FIRE FIGHTING INFORMATION:

VX is combustible.

The agent may burn but does not ignite readily.

Fire may produce irritating, corrosive, and/or toxic gases.

For small fires, use dry chemical, carbon dioxide, or water spray.

For large fires, use dry chemical, carbon dioxide, alcohol-resistant foam, or water spray. Move containers from the fire area if it is possible to do so without risk to personnel. Dike fire control water for later disposal; do not scatter the material.

Avoid methods that will cause splashing or spreading.

For fire involving tanks or car/trailer loads, fight the fire from maximum distance or use unmanned hose holders or monitor nozzles. Do not get water inside containers. Cool containers with flooding quantities of water until well after the fire is out. Withdraw immediately in case of rising sound from venting safety devices or discoloration of tanks. Always stay away from tanks engulfed in fire.

Runoff from fire control or dilution water may be corrosive and/or toxic, and it may cause pollution.

If the situation allows, control and properly dispose of run-off (effluent).

INITIAL ISOLATION AND PROTECTIVE ACTION DISTANCES:

If a tank, rail car, or tank truck is involved in a fire, isolate it for 0.5 mi (800 m) in all directions; also, consider initial evacuation for 0.5 mi (800 m) in all directions.

Small spills (involving the release of approximately 52.83 gallons (200 liters) or less), when VX is used as a weapon

First isolate in all directions: 100 ft (30 m).

Then protect persons downwind during the day: 0.1 mi (0.2 km).

Then protect persons downwind during the night: 0.1 mi (0.2 km).

Large spills (involving quantities greater than 52.83 gallons (200 liters)), when VX is used as a weapon

First isolate in all directions: 200 ft (60 m).

Then protect persons downwind during the day: 0.4 mi (0.7 km).

Then protect persons downwind during the night: 0.6 mi (1.0 km).

PHYSICAL DANGERS:

VX is persistent in the environment.

Vapors are heavier than air. They will spread along the ground and collect and stay in poorly-ventilated, low-lying, or confined areas (e.g., sewers, basements, and tanks).

Hazardous concentrations may develop quickly in enclosed, poorly-ventilated, or low-lying areas. Keep out of these areas. Stay upwind.

NFPA 704 Signal:

Health: 4

Flammability: 1

Reactivity: 0

Special:

Signs/Symptoms

TIME COURSE: Exposure to nerve agents may be rapidly fatal. Eye exposure: Liquid VX produces health effects within seconds to minutes; larger exposures may cause death within 1 to 10 minutes. Ingestion exposure: No information is available on the time course of effects following ingestion of VX. Inhalation exposure: Inhaled VX produces health effects within seconds to minutes; larger exposures may cause death within 1 to 10 minutes. Skin exposure: Liquid VX may produce health effects within minutes. Health effects from mild to moderate exposure may be delayed up to 18 hours; larger exposures may cause death within minutes to hours.

EFFECTS OF SHORT-TERM (LESS THAN 8-HOURS) EXPOSURE: Nerve agents cause the same health effects regardless of the route of exposure. Initial effects depend on the dose and route of exposure. Nerve agents interfere with the normal functioning of the nervous system. Skeletal muscles, certain organs of the body, and the central nervous system (CNS) may all be affected by exposure to the nerve agent.

EYE EXPOSURE:

Contracted or pinpoint pupils (miosis), redness of the membranes (conjunctiva), pain in and around the eye, dim and/or blurred vision, a sensation of pressure with heaviness, and reflex nausea and vomiting (emesis).

Effects are usually local, occurring from direct contact with nerve agent vapor, aerosol, or liquid; but exposure by other routes can also affect the eyes.

INGESTION EXPOSURE:

Nausea, vomiting (emesis), diarrhea, abdominal pain, and cramping.

INHALATION EXPOSURE:

Mild to moderate: Contracted or pinpoint pupils (miosis), runny nose (rhinorrhea), narrowing of the large airways (bronchoconstriction), fluid build up within the airways of the lungs, and slight to moderate difficulty breathing or shortness of breath (dyspnea).

Severe: In addition to the symptoms described above, there can be loss of consciousness; seizures; muscular twitching (fasciculations); floppy (flaccid) paralysis; increased fluid build up within the airways and within the digestive tract, resulting in secretions from the nose and mouth; cessation of breathing (apnea); and death.

SKIN EXPOSURE:

Mild to moderate: Health effects may be immediate or may be delayed up to 18 hours. Profuse sweating (diaphoresis) and muscular twitching (fasciculations) at the site of contact, nausea, vomiting (emesis), diarrhea, and weakness (malaise).

Severe: Health effects may appear quickly; 2 to 30 minutes post-exposure. In addition to the symptoms described above, there can be loss of consciousness; seizures; muscular twitching (fasciculations); floppy (flaccid) paralysis; increased fluid build up within the airways and within the digestive tract, resulting in secretions from the nose and mouth; cessation of breathing (apnea), and death.

Decontamination.

INTRODUCTION: The purpose of decontamination is to make an individual and/or their equipment safe by physically removing toxic substances quickly and effectively. Care should be taken during decontamination, because absorbed agent can be released from clothing and skin as a gas. Your Incident Commander will provide you with decontaminants specific for the agent released or the agent believed to have been released.

DECONTAMINATION CORRIDOR: The following are recommendations to protect the first responders from the release area:

Position the decontamination corridor upwind and uphill of the hot zone.

The warm zone should include two decontamination corridors. One decontamination corridor is used to enter the warm zone and the other for exiting the warm zone into the cold zone. The decontamination zone for exiting should be upwind and uphill from the zone used to enter.

Decontamination area workers should wear appropriate PPE. See the PPE section of this card for detailed information.

A solution of detergent and water (which should have a pH value of at least 8 but should not exceed a pH value of 10.5) should be available for use in decontamination procedures. Soft brushes should be available to remove contamination from the PPE.

Labeled, durable 6-mil polyethylene bags should be available for disposal of contaminated PPE.

INDIVIDUAL DECONTAMINATION: The following methods can be used to decontaminate an individual:

Decontamination of First Responder:

Begin washing PPE of the first responder using soap and water solution and a soft brush. Always move in a downward motion (from head to toe). Make sure to get into all areas, especially folds in the clothing. Wash and rinse (using cold or warm water) until the contaminant is thoroughly removed.

Remove PPE by rolling downward (from head to toe) and avoid pulling PPE off over the head. Remove the SCBA after other PPE has been removed.

Place all PPE in labeled durable 6-mil polyethylene bags.

Decontamination of Patient/Victim:

Remove the patient/victim from the contaminated area and into the decontamination corridor.

Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag.

Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds.

Cover the patient/victim to prevent shock and loss of body heat.

Move the patient/victim to an area where emergency medical treatment can be provided.

First Aid.

GENERAL INFORMATION: Initial treatment consists of repeated administration of antidotes and supportive measures.

ANTIDOTE: Atropine and pralidoxime chloride (2-PAM Cl) are antidotes for nerve agent toxicity; however, 2-PAM Cl must be administered within minutes to a few hours (depending on the agent) following exposure to be effective. There is also generally no benefit in giving more than three injections of 2-PAM Cl. Atropine should be administered every 5 to 10 minutes until secretions begin to dry up. If the military Mark I kits containing autoinjectors are available, they provide the best way to administer the antidotes to healthy adults. One autoinjector automatically delivers 2 mg atropine and the other automatically delivers 600 mg 2-PAM Cl. If the Mark I kit is unavailable, or the patient/victim is not an otherwise healthy adult, administer antidotes as described below:

Infant (0–2 yrs), for mild to moderate physical findings, including localized sweating, muscular twitching (fasciculations), nausea, vomiting, weakness, and shortness of breath (dyspnea); administer Atropine at 0.05 mg/kg IM; 2-PAM Cl at 15 mg/kg IM.

Infant (0–2 yrs), for severe physical findings, including unconsciousness, convulsions, cessation of breathing (apnea), and floppy (flaccid) paralysis; administer Atropine at 0.1 mg/kg IM; 2-PAM Cl at 25 mg/kg IM.

Child (2–10 yrs), for mild to moderate physical findings, including localized sweating, muscular twitching (fasciculations), nausea, vomiting, weakness, and shortness of breath (dyspnea); administer Atropine at 1 mg/kg IM; 2-PAM Cl at 15 mg/kg IM.

Child (2–10 yrs), for severe physical findings, including unconsciousness, convulsions, cessation of breathing (apnea), and floppy (flaccid) paralysis; administer Atropine at 2 mg/kg IM; 2-PAM Cl at 25 mg/kg IM.

Adolescent (> 10 yrs), for mild to moderate physical findings, including localized sweating, muscular twitching (fasciculations), nausea, vomiting, weakness, and shortness of breath (dyspnea); administer Atropine at 2 mg/kg IM; 2-PAM Cl at 15 mg/kg IM.

Adolescent (> 10 yrs), for severe physical findings, including unconsciousness, convulsions, cessation of breathing (apnea), and floppy (flaccid) paralysis; administer Atropine at 4 mg IM; 2-PAM Cl at 25 mg/kg IM.

Adult, for mild to moderate physical findings, including localized sweating, muscular twitching (fasciculations), nausea, vomiting, weakness, and shortness of breath (dyspnea); administer Atropine at 2 to 4 mg IM; 2-PAM Cl at 600 mg IM.

Adult, for severe physical findings, including unconsciousness, convulsions, cessation of breathing (apnea), and floppy (flaccid) paralysis; administer Atropine at 6 mg IM; 2-PAM Cl at 1800 mg IM.

Elderly, frail for mild to moderate physical findings, including localized sweating, muscular twitching (fasciculations), nausea, vomiting, weakness, and shortness of breath (dyspnea); administer Atropine at 1 mg IM; 2-PAM Cl at 10 mg/kg IM.

Elderly, frail for severe physical findings, including unconsciousness, convulsions, cessation of breathing (apnea), and floppy (flaccid) paralysis; administer Atropine at 2 to 4 mg IM; 2-PAM Cl at 25 mg/kg IM.

Assisted ventilation should be started after administration of antidotes for severe exposures.

Repeat atropine (2 mg IM for adults or 0.05 to 0.1 mg/kg for children) at 5 to 10 minute intervals until secretions have diminished and breathing is comfortable or airway resistance has returned to near normal.

EYE:

Immediately remove the patient/victim from the source of exposure.

Often the first physical finding of minimal symptomatic exposure to nerve agent vapor is markedly constricted pupils (miosis); however, if this is the only physical finding of nerve agent exposure, do not administer antidotes but follow the instructions below.

When exposed to liquid nerve agent, immediately flush the eyes with water for about 5 to 10 minutes by tilting the head to the side, pulling the eyelids apart with fingers, and pouring water slowly into the eyes.

When exposed to nerve agent vapor, there is no need to flush the eyes.

Do not cover eyes with bandages.

Changes in the eye can lead to nausea and vomiting without necessarily being a sign of systemic exposure. However, if eye pain, nausea, or vomiting are seen in combination with any other physical findings of nerve agent poisoning, administer antidotes atropine and 2-PAM Cl as directed.

Seek medical attention immediately.

INGESTION:

Immediately remove the patient/victim from the source of exposure.

Ensure that the patient/victim has an unobstructed airway.

Do not induce vomiting (emesis).

Administer nothing by mouth (NPO).

If the patient/victim’s condition can be evaluated within 30 minutes after ingestion, in a hospital setting, consider gastric lavage. Gastric contents should be considered potentially hazardous and should be quickly isolated.

Be alert to physical findings of systemic exposure, and administer antidotes as required.

Maintain records of all injections given.

Seek medical attention immediately.

INHALATION:

Immediately remove the patient/victim from the source of exposure.

In cases of moderate to severe exposure, antidotes alone will not provide effective treatment, and ventilatory support is essential.

Evaluate respiratory function and pulse.

Ensure that the patient/victim has an unobstructed airway.

Assist with ventilation as required. Do not provide mouth-to-mouth resuscitation. Contact with off-gassed vapor or with liquid agent may occur.

If shortness of breath occurs or breathing is difficult (dyspnea), administer oxygen.

Suction secretions from the nose, mouth, and respiratory tract.

Marked resistance to ventilation is expected due to bronchial constriction and spasm. Resistance lessens after administration of atropine.

Ventilatory distress is a physical finding of systemic exposure and requires antidote administration.

Maintain records of all injections given.

Seek medical attention immediately.

SKIN:

Immediately remove the patient/victim from the source of exposure.

Some nerve agents may remain in the hair or clothing and should be decontaminated if that was not previously done. See the decontamination section of this card.

Skin exposure to liquid nerve agents will not necessarily result in systemic exposure if the site of exposure is decontaminated promptly. Before administering nerve agent antidotes, observe the site of exposure for localized sweating and muscular twitching. If these physical findings appear, administer antidotes, otherwise careful observation is all that is needed.

Maintain records of all injections given.

Seek medical attention immediately.

See ATSDR Medical Management Guidelines for Nerve Agents for more detailed recommendations, https://www.atsdr.cdc.gov/MHMI/mmg166.pdf.

Long-Term Implications

MEDICAL TREATMENT: Electrocardiogram (ECG), and adequacy of respiration and ventilation, should be monitored. Supplemental oxygenation, frequent suctioning of secretions, insertion of a tube into the trachea (endotracheal intubation), and assisted ventilation may be required. Diazepam (5 to 10 mg in adults and 0.2 to 0.5 mg/kg in children) may be used to control convulsions. Lorazepam or other benzodiazepines may be used, but barbiturates, phenytoin, and other anticonvulsants are not effective. Administration of atropine (if not already given) should precede the administration of benzodiazepines in order to best control seizures. Patients/victims who have inhalation exposure and who complain of chest pain, chest tightness, or cough should be observed and examined periodically for 6 to 12 hours to detect delayed-onset inflammation of the large airways (bronchitis), inflammatory lung disease (pneumonia), accumulation of fluid in the lungs (pulmonary edema), or respiratory failure.

DELAYED EFFECTS OF EXPOSURE: Patients/victims who have severe exposure should be evaluated for persistent central nervous system (CNS) effects.

EFFECTS OF CHRONIC OR REPEATED EXPOSURE: Limited data are available on long-term exposures of humans to VX. The available data do

not suggest that VX is a human carcinogen, reproductive toxin or developmental toxin. Chronic or repeated exposure to VX may result in characteristic nervous, digestive, locomotory, visual and cardiovascular symptoms.

On-Site Fatalities.

INCIDENT SITE:

Consult with the Incident Commander regarding the agent dispersed, dissemination method, level of PPE required, location, geographic complications (if any), and the approximate number of remains.

Coordinate responsibilities and prepare to enter the scene as part of the evaluation team along with the FBI HazMat Technician, local law enforcement evidence technician, and other relevant personnel.

Begin tracking remains using waterproof tags.

RECOVERY AND ON-SITE MORGUE:

Wear PPE until all remains are deemed free of contamination.

Establish a preliminary (holding) morgue.

Gather evidence, and place it in a clearly labeled impervious container. Hand any evidence over to the FBI.

Remove and tag personal effects.

Perform a thorough external evaluation and a preliminary identification check.

See the Decontamination section for decontamination procedures.

Decontaminate remains before they are removed from the incident site.

ENVIRONMENT/SPILLAGE DISPOSAL: The following methods can be used to decontaminate the environment/spillage disposal:

Do not touch or walk through the spilled agent if at all possible. However, if you must, personnel should wear the appropriate PPE during environmental decontamination. See the PPE section of this card for detailed information.

Keep combustibles (e.g., wood, paper, and oil) away from the spilled agent.

Use water spray to reduce vapors or divert vapor cloud drift. Avoid allowing water runoff to contact the spilled agent.

Do not direct water at the spill or the source of the leak.

Stop the leak if it is possible to do so without risk to personnel, and turn leaking containers so that gas rather than liquid escapes.

Prevent entry into waterways, sewers, basements, or confined areas.

Isolate the area until gas has dispersed.

Ventilate the area.

EQUIPMENT: Agents can seep into the crevices of equipment making it dangerous to handle. The following methods can be used to decontaminate equipment:

Not established/determined

Agent Properties

Chemical Formula:

$C_{11}H_{26}NO_2PS$

Aqueous solubility:

Slightly soluble

Boiling Point:
568.4 °F (298 °C)
Density:
Liquid: 1.008 g/mL at 68 °F (20 °C)
Vapor: 9.2 (air = 1)
Flammability:
Combustible
Flashpoint:
318.2 °F (159 °C)
Ionization potential:
Not established/determined
Log $K_{benzene\text{-}water}$:
Not established/determined
Log K_{ow} (estimated):
Not established/determined
Melting Point:
Below -59.8 °F (-51 °C)
Molecular Mass:
267.37
Soluble In:
All solvents
Specific Gravity:
1.008
Vapor Pressure:
0.0007 mm Hg at 77 °F (25 °C)
Volatility:
10.5 mg/m^3 at 77 °F (25 °C)

CHAPTER 5
MUSTARD (BLISTER) AGENTS

Introduction

Mustard agents are usually classified as "blistering agents" owing to the similarity of the wounds caused by these substances resembling burns and blisters. However, since mustard agents also cause severe damage to the eyes, respiratory system and internal organs, they should preferably be described as «blistering and tissue-injuring agents». Normal mustard agent, bis-(2-chloroethyl) sulphide, reacts with a large number of biological molecules. The effect of mustard agent is delayed and the first symptoms do not occur until 2–24 hours after exposure.

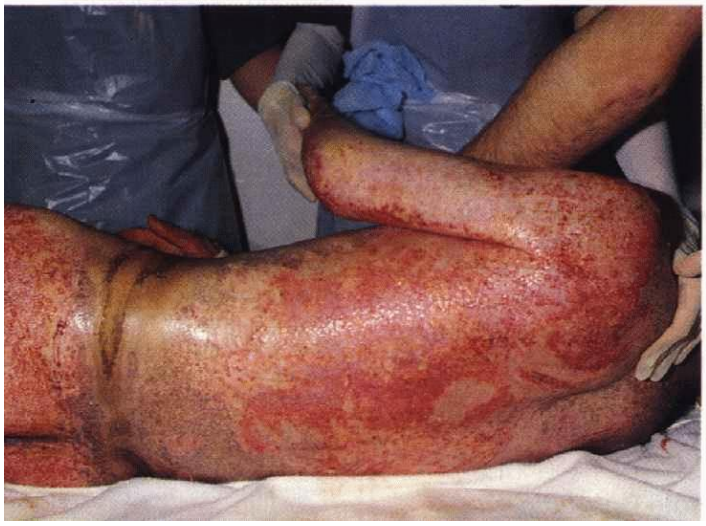

Figure 21 — One of the Iranian soldiers who were being treated for mustard agent burns in a Swedish hospital. Injuries from mustard agent often require long periods of care. The photo shows extensive injury, several weeks old, which is now starting to heal

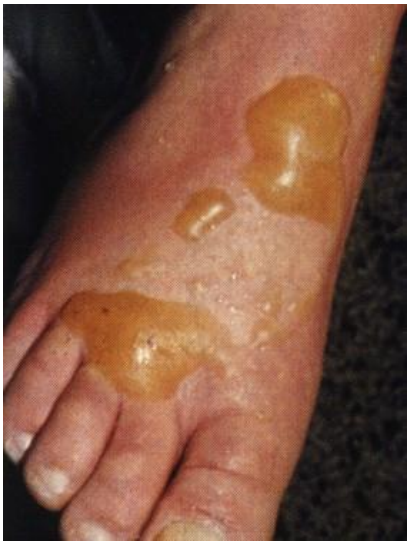

Figure 22 — A Baltic fisherman with a relatively fresh mustard agent injury. Inflammation and fluid-filled blisters can be seen on the foot

Mustard agents are usually classified as «blistering agents» owing to the similarity of the wounds caused by these substances resembling burns and blisters. However, since mustard agents also cause severe damage to the eyes, respiratory system and internal organs, they should preferably be described as «blistering and tissue-injuring agents». Normal mustard agent, bis-(2-chloroethyl) sulphide, reacts with a large number of biological molecules. The effect of mustard agent is delayed and the first symptoms do not occur until 2-24 hours after exposure.

Mustard agent was produced for the first time in 1822 but its harmful effects were not discovered until 1860. Mustard agent was first used as a CW agent during the latter part of the First World War and caused lung and eye injuries to a very large number of soldiers. Many of them still suffered pain 30–

40 years after they had been exposed, mainly as a result of injuries to the eyes and chronic respiratory disorders.

During the war between Iran and Iraq in 1979–1988, Iraq used large quantities of chemical agents. About 5 000 Iranian soldiers have been reported killed, 10–20 per cent by mustard agent. In addition, there were 40 000 to 50 000 injured. A typical result of warfare with mustard agent is that the medical system is overloaded with numerous victims who require long and demanding care.

Incidents are still occurring annually in the neighbourhood of Sweden where people risk injury from mustard agent. This largely involves fishermen who are exposed to mustard agent brought to the surface by fishing nets. The background is found in the dumping of chemical weapons after the Second World War in waters off the Danish and Swedish coasts. Many fishing ports in south Sweden and Denmark have resources to care for injured people and to decontaminate equipment contaminated by mustard agent. Certain resources are also available on the fishing vessels.

Mustard agent is very simple to manufacture and can therefore be a «first choice» when a country decides to build up a capacity for chemical warfare.

Apart from mustard agent, there are also several other closely related compounds which have been used as chemical weapons. During the 1930's, several reports were published on the synthesis of nitrogen mustard agent and its remarkable blistering effect. The mechanism of action and symptoms largely agree with those described for mustard agent. Germans and Americans started the military production of nitrogen mustard agent in 1941 and 1943, respectively, whereas the development in England was abandoned following an explosion. There is no verified use of nitrogen mustard agents as chemical weapons and their usefulness is restricted by these types of agents being unsuitable for storage.

Physical and Chemical Properties

In its pure state, mustard agent is colourless and almost odourless. The name was given to mustard agent as a result of an earlier production method which yielded an impure mustard-smelling product. Mustard agent is also claimed to have a characteristic smell similar to rotten onions. However, the sense of smell is dulled after only a few breaths so that the smell can no longer be distinguished. In addition, mustard agent can cause injury to the respiratory system in concentrations which are so low that the human sense of smell cannot distinguish them.

At room temperature, mustard agent is a liquid with low volatility and is very stable during storage. The melting-point for pure mustard agent is 14.4 °C. In order to be able to effectively use mustard agent at lower temperatures, it has been mixed with lewisite in some types of ammunition in a ratio of 2:3. This mixture has a freezing-point of -26 °C. During the Second World War, a form of mustard agent with high viscosity was manufactured by means of the addition of a polymer. This is the first known example of a thickened CW agent.

Mustard agent can easily be dissolved in most organic solvents but has poor solubility in water. In aqueous solutions, mustard agent decomposes into non-poisonous products by means of hydrolysis. This reaction is catalyzed by alkali. However, only dissolved mustard agent reacts, which means that the decomposition proceeds very slowly. Bleaching-powder and chloramines, however, react violently with mustard agent, whereupon non-poisonous oxidation products are formed. Consequently, these substances are used for the decontamination of mustard agent.

Physical Properties of Mustard Agent

Molecular weight	159.1
Density g/cm³ at 25 °C	1.27
Boiling-point °C	217
Melting-point °C	14
Vapour pressure mm Hg at 25 °C	0.11
Volatility mg/m³ at 25 °C	900
Solubility in water % at 20 °C	0.06

Mechanism of Action

The toxic effects of mustard agent depend on its ability to covalently bind to other substances. The chlorine atom is spiked off the ethyl group and the mustard agent is transferred to a reactive sulphonium ion. This ion can bind to a large number of different biological molecules. Most of all it binds to nucleophiles such as nitrogen in the base components of nucleic acids and sulphur in SH-groups in proteins and peptides. Since mustard agent contains two "reactive groups", it can also form a bridge between or within molecules. Mustard agent can destroy a large number of different substances in the cell by means of alkylation and thereby influence numerous processes in living tissue.

Toxicity of Mustard Agent

LCt^{50} (mg*min/m³)	inhalation	1,500
LD^{50} (mg/kg)	skin exposure	10,000
Smallest blister-causing dose on skin (mg)		0.02

Symptoms

In the form of gas or liquid, mustard agent attacks the skin, eyes, lungs and gastro-intestinal tract. Internal organs may also be injured, mainly blood-generating organs, as a result of mustard agent being taken up through the skin or lungs and transported into the body. The delayed effect is a characteristic of

mustard agent. Mustard agent gives no immediate symptoms upon contact and consequently a delay of between two and twenty-four hours may occur before pain is felt and the victim becomes aware of what has happened. By then cell damage has already been caused.

Symptoms of mustard agent poisoning extend over a wide range. Mild injuries consist of aching eyes with abundant flow of tears, inflammation of the skin, irritation of the mucous membrane, hoarseness, coughing and sneezing. Normally, these injuries do not require medical treatment. Severe injuries which are incapacitating and require medical care may involve eye injuries with loss of sight, the formation of blisters on the skin, nausea, vomiting and diarrhoea together with severe respiration difficulty.

Acute mortality arising from exposure to mustard agent is low. The dose needed to directly kill a person upon inhalation is, e.g., about 50 times larger than the dose giving acute mortality upon poisoning with the nerve agent soman. People who die after exposure to mustard agent usually do so after a few days up to one or more weeks.

Minor skin damage may be caused by mustard agent in the gaseous state whereas the most severe injuries are caused after contact with liquid mustard agent. Skin damage first appears as a painful inflammation. Depending on the level of exposure, the injury may develop into pigmentation, which flakes-off after a couple of weeks, small surface blisters or deep liquid-filled blisters with subsequent skin necrosis. In extreme cases, the skin necrosis may be so comprehensive that no blisters occur. Skin injuries are more severe in humid and warm climates. Similarly, the injuries will be more severe where the skin is moist and warm, e.g., in the groin and armpits.

Experience has shown that even extremely extensive skin damage, 80–90 %, can be cured if the patient is kept free of infection. However, injuries to the skin require a very long period of recuperation, much longer than thermal burns, and may require care and plastic surgery over a period of several months.

Injury to the eyes appear initially as irritation with eye inflammation and a strong flow of tears. Depending on exposure, the symptoms thereafter may successively develop to sensitivity to light, swollen eyelids, and injury to the cornea. Severe damage to the eye may lead to the total loss of vision. Victims suffering damage to the eyes may encounter problems persisting up to 30-40 years following exposure.

The most common cause of death as a result of mustard agent poisoning is complications after lung injury caused by inhalation of mustard agent. Lung injuries become apparent some hours after exposure and will first appear as a pressure across the chest, sneezing and hoarseness. Severe coughing and respiration difficulties caused by pulmonary oedema will gradually occur and after a couple of days, a «chemical pneumonia» may develop. Most of the chronic and late effects are also caused by lung injuries.

The effect on inner organs which is most pronounced is injury to the bone marrow, spleen and lymphatic tissue. This may cause a drastic reduction in the number of white blood cells 5-10 days after exposure, a condition very similar to that after exposure to radiation. This reduction of the immune defence will complicate the already large risk of infection in people with severe skin and lung injuries.

Antidotes and Methods of Treatment

There is no treatment or antidote which can affect the basic cause of mustard agent injury. Instead, efforts must be made to treat the symptoms. By far the most important measure is to rapidly and thoroughly decontaminate the patient and thereby prevent further exposure. This decontamination will also decrease the risk of exposure to staff. Clothes are removed, the skin is decontaminated with a suitable decontaminant and washed with soap and water. If hair is suspected to be contaminated then it must be shaved off. Eyes are rinsed with water or a physiological salt solution for at least five minutes.

In medical treatment, efforts are made to control infections by means of antibiotics. Pain can be eased by local anesthetics. After skin injuries have healed, it may be necessary to introduce plastic surgery. Lung injuries are treated with bronchodilatory treatment. Medicine to relieve coughing and also cortisone preparations may be used. Eye injuries are treated locally with painkillers and with antibiotics if required. Despite treatment, inflammation and light sensitivity may remain for long periods.

Modern knowledge on the mechanisms behind mustard agent injuries may lead mainly to new ways of treatment. The first step, alkylation, takes place extremely rapidly and is probably very difficult to influence. Future treatment may concentrate on suppressing and alleviating the development of symptoms and thereby improve the opportunities for good recovery.

Types of Injury Caused by Mustard Agent

It is impossible to identify a single mechanism for the damage caused by mustard agent. However, two possible important mechanisms can be mentioned where the first step in both is the formation of a reactive sulphonium ion. One such mechanism is the bonding of mustard agent to the base compounds in DNA (alkylation). The bonding may induce breakages of strands and the formation of bridges between the two strands in the DNA molecule. Bridges of this kind prevent DNA from functioning normally during cell division which may lead to severe injury and possibly cell mortality. Damage to the DNA may also lead to mutations and disturbance to the natural repair mechanisms of DNA. The influence on DNA can cause the increased frequency of cancer observed after exposure to mustard agent.

The other mechanism of action is interaction between mustard agent and intracellular glutathion. Glutathion is a small peptide molecule which, among

other things, takes care of the free radicals formed during cell respiration. If too large an amount of glutathion is bound by mustard agent, then the regulation of these free radicals no longer functions. Since free radicals are extremely toxic, this may lead to a number of processes in the cell being severely disturbed.

Mustard agent can also bind to different proteins in the cell. However, it is not known how much this contributes to the injuries caused. The binding takes place at the functional groups, e.g., the sulphydryl or amino groups. If the binding is made to, for example, the active site of enzymes, then their activity is inhibited which could lead to metabolic disorders. If, on the other hand, membrane proteins are bound, the result can be a modified uptake of substances and the inner environment of the cell will become disturbed.

LEWISITE (L)

Common Names:

Chlorovinylarsine dichloride

Dichloro(2-chlorovinyl)arsine

Agent Characteristics

APPEARANCE: Oily liquid with a range of colors from colorless to violet-black, green, amber, or dark brown.

DESCRIPTION: Lewisite is an extremely toxic, arsenic-containing blister agent (vesicant) that affects the lungs and causes whole-body (systemic) effects. It has an odor of geraniums. It was developed as a potential chemical warfare agent (military designation, L), but has not been used on the battlefield. Exposure to large amounts can be fatal. Pain and irritation from exposure to either liquid or vapor lewisite are immediate, and early tissue destruction is more obvious than after exposure to mustard. Lewisite causes burning pain in the respiratory tract at a concentration that cannot be detected by odor.

METHODS OF DISSEMINATION:

Indoor Air: Lewisite can be released into indoor air as a liquid spray (aerosol) or as a vapor.

Water: Lewisite can contaminate water.

Food: Lewisite can contaminate food.

Outdoor Air: Lewisite can be released into outdoor air as a liquid spray (aerosol) or as a vapor.

Agricultural: If lewisite is released into the air as a liquid spray (aerosol), it has the potential to contaminate agricultural products. If lewisite is released as a vapor, it is highly unlikely to contaminate agricultural products.

ROUTES OF EXPOSURE: Lewisite can be absorbed into the body by inhalation, ingestion, skin contact, or eye contact. Inhalation is an important route of exposure. Ingestion is an uncommon route of exposure.

Personal Protective Equipment.

GENERAL INFORMATION: First Responders should use a NIOSH-certified Chemical, Biological, Radiological, Nuclear (CBRN) Self Contained

Breathing Apparatus (SCBA) with a Level A protective suit when entering an area with an unknown contaminant or when entering an area where the concentration of the contaminant is unknown. Level A protection should be used until monitoring results confirm the contaminant and the concentration of the contaminant.

NOTE: Safe use of protective clothing and equipment requires specific skills developed through training and experience.

LEVEL A: (RED ZONE): Select when the greatest level of skin, respiratory, and eye protection is required. This is the maximum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than the AEGL-2.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A Totally-Encapsulating Chemical Protective (TECP) suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, and a hard hat worn under the TECP suit are optional items.

LEVEL B: (RED ZONE): Select when the highest level of respiratory protection is necessary but a lesser level of skin protection is required. This is the minimum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than AEGL-2. It differs from Level A in that it incorporates a non-encapsulating, splash-protective, chemical-resistant splash suit that provides Level A protection against liquids but is not airtight.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL C: (YELLOW ZONE): Select when the contaminant and concentration of the contaminant are known and the respiratory protection criteria factors for using Air Purifying Respirators (APR) or Powered Air Purifying Respirators (PAPR) are met. This level is appropriate when decontaminating patient/victims.

A NIOSH-certified CBRN tight-fitting APR with a canister-type gas mask or CBRN PAPR for air levels greater than AEGL-2.

A NIOSH-certified CBRN PAPR with a loose-fitting face-piece, hood, or helmet and a filter or a combination organic vapor, acid gas, and particulate cartridge/filter combination or a continuous flow respirator for air levels greater than AEGL-1.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Escape mask, face shield, coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL D: (GREEN ZONE): Select when the contaminant and concentration of the contaminant are known and the concentration is below the appropriate occupational exposure limit or less than AEGL-1 for the stated duration times.

Limited to coveralls or other work clothes, boots, and gloves.

Emergency Response

CHEMICAL DANGERS:

Lewisite hydrolyzes in acidic medium to form hydrochloric acid and non-volatile (solid) chlorovinylarsenous oxide, a less potent blister agent than lewisite. See the emergency response cards for hydrochloric acid and chlorovinylarsenous oxide.

Lewisite hydrolyzes in basic media, as in decontamination with alcoholic, caustic, or carbonate solution, to form acetylene and trisodium arsenate. See the emergency response cards for acetylene and trisodium arsenate.

Decontamination wash water (effluent) will contain toxic arsenic. See the emergency response card for arsenic.

Contact with metals may evolve flammable hydrogen gas.

EXPLOSION HAZARDS:

When heated, vapors may form explosive mixtures with air, presenting an explosion hazard indoors, outdoors, and in sewers.

Containers may explode when heated.

FIRE FIGHTING INFORMATION:

Lewisite is combustible.

The agent may burn but does not ignite readily.

Fire may produce irritating, corrosive, and/or toxic gases.

For small fires, use dry chemical, carbon dioxide, or water spray.

For large fires, use dry chemical, carbon dioxide, alcohol-resistant foam, or water spray. Move containers from the fire area if it is possible to do so without risk to personnel. Dike fire control water for later disposal; do not scatter the material.

Avoid methods that will cause splashing or spreading.

For fire involving tanks or car/trailer loads, fight the fire from maximum distance or use unmanned hose holders or monitor nozzles. Do not get water inside containers. Cool containers with flooding quantities of water until well after the fire is out. Withdraw immediately in case of rising sound from venting safety devices or discoloration of tanks. Always stay away from tanks engulfed in fire.

Runoff from fire control or dilution water may be corrosive and/or toxic, and it may cause pollution.

If the situation allows, control and properly dispose of run-off (effluent).

INITIAL ISOLATION AND PROTECTIVE ACTION DISTANCES:

If a tank, rail car, or tank truck is involved in a fire, isolate it for 0.5 mi (800 m) in all directions; also, consider initial evacuation for 0.5 mi (800 m) in all directions.

Small spills (involving the release of approximately 52.83 gallons (200 liters) or less), when lewisite (L) is used as a weapon

First isolate in all directions: 100 ft (30 m).

Then protect persons downwind during the day: 0.1 mi (0.2 km).

Then protect persons downwind during the night: 0.2 mi (0.4 km).

Large spills (involving quantities greater than 52.83 gallons (200 liters)), when lewisite (L) is used as a weapon

First isolate in all directions: 300 ft (90 m).

Then protect persons downwind during the day: 0.6 mi (1.0 km).

Then protect persons downwind during the night: 1.1 mi (1.8 km).

PHYSICAL DANGERS:

Vapors are heavier than air. They will spread along the ground and collect and stay in poorly-ventilated, low-lying, or confined areas (e.g., sewers, basements, and tanks).

Hazardous concentrations may develop quickly in enclosed, poorly-ventilated, or low-lying areas. Keep out of these areas. Stay upwind.

Lewisite remains a liquid at low temperatures and is persistent in colder climates.

NFPA 704 Signal:

Health: 4

Flammability: 1

Reactivity: 1

Special:

SAMPLING AND ANALYSIS:

OSHA: Not established/determined

NIOSH: Not established/determined

Signs/Symptoms

TIME COURSE: Lewisite produces health effects within seconds to minutes after exposure. Eye exposure: Lewisite produces pain and/or irritation within seconds to minutes of exposure. Redness occurs within 15 to 30 minutes following exposure to liquid lewisite. Inhalation exposure: Lewisite produces immediate burning pain; this may cause exposed patients/victims to seek protec-

tion and limit their exposure. Skin exposure: Lewisite produces immediate stinging pain; redness (erythema) within 15 to 30 minutes, with pain and itching for 24 hours; and blistering (vesication) within 12 hours, with pain for 2 to 3 days. Blistering begins within hours following exposure, but the full extent of blistering does not occur for 12 to 18 hours. The blister begins small in the center of a red area, and then expands to include the entire area of inflammation. Exposure to liquid lewisite causes skin lesions to occur sooner than does exposure to vapor. Lewisite is absorbed by the skin within 3 to 5 minutes following exposure, and may result in shock.

EFFECTS OF SHORT-TERM (LESS THAN 8-HOURS) EXPOSURE: Lewisite locally damages the skin, eyes, and airways by direct contact. After absorption into the body, it may cause more widespread health effects. "Lewisite shock" is seen after exposure to large amounts of lewisite. It is caused when absorbed lewisite damages the smallest blood vessels (capillaries) of the body. Damage to the capillaries results in the leakage of blood components (proteins and plasma) into the surrounding tissues and a decrease in the volume of circulating blood (hypovolemia). The decrease in the volume of circulating blood may injure the kidneys and cause seriously low blood pressure (hypotension).

EYE EXPOSURE:

Mild to moderate: Immediate stinging and burning pain and strong irritation, tear production (lacrimation), spasmodic blinking (blepharospasm), and swelling and fluid accumulation (edema) in the membranes and eyelids.

Eyes are likely to swell shut, reducing further exposure to lewisite vapor.

Severe: Blistering (vesication) and scarring of the cornea, rupture (perforation) of the eye, and blindness.

INGESTION EXPOSURE:

Nausea and vomiting (emesis).

INHALATION EXPOSURE:

Mild to moderate: Irritation of the nose and lower airways, immediate burning pain, violent sneezing, nosebleed (epistaxis), sinus pain, inflammation of the voice box (laryngitis), cough, and difficulty breathing or shortness of breath (dyspnea).

Severe: Inflammation of the lungs (pneumonitis), accumulation of fluid in the lungs (pulmonary edema), respiratory failure, and death.

SKIN EXPOSURE:

Mild to moderate: Immediate stinging and burning pain or irritation, redness (erythema), blistering (vesication) with pain, and itching (pruritus).

Severe: Severe blistering (vesication) and severe burns.

Decontamination

INTRODUCTION: The purpose of decontamination is to make an individual and/or their equipment safe by physically removing toxic substances quickly and effectively. Care should be taken during decontamination, because absorbed agent can be released from clothing and skin as a gas. Your Incident Commander will provide you with decontaminants specific for the agent released or the agent believed to have been released.

DECONTAMINATION CORRIDOR: The following are recommendations to protect the first responders from the release area:

Position the decontamination corridor upwind and uphill of the hot zone.

The warm zone should include two decontamination corridors. One decontamination corridor is used to enter the warm zone and the other for exiting the warm zone into the cold zone. The decontamination zone for exiting should be upwind and uphill from the zone used to enter.

Decontamination area workers should wear appropriate PPE. See the PPE section of this card for detailed information.

A solution of detergent and water (which should have a pH value of at least 8 but should not exceed a pH value of 10.5) should be available for use in decontamination procedures. Soft brushes should be available to remove contamination from the PPE.

Labeled, durable 6-mil polyethylene bags should be available for disposal of contaminated PPE.

INDIVIDUAL DECONTAMINATION: The following methods can be used to decontaminate an individual:

Decontamination of First Responder:

Begin washing PPE of the first responder using soap and water solution and a soft brush. Always move in a downward motion (from head to toe). Make sure to get into all areas, especially folds in the clothing. Wash and rinse (using cold or warm water) until the contaminant is thoroughly removed.

Remove PPE by rolling downward (from head to toe) and avoid pulling PPE off over the head. Remove the SCBA after other PPE has been removed.

Place all PPE in labeled durable 6-mil polyethylene bags.

Decontamination of Patient/Victim:

Remove the patient/victim from the contaminated area and into the decontamination corridor.

Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag.

Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds.

Cover the patient/victim to prevent shock and loss of body heat.

Move the patient/victim to an area where emergency medical treatment can be provided.

First Aid.

GENERAL INFORMATION: Rapid decontamination (within minutes of exposure) is the only way to limit injury. Initial treatment is primarily supportive.

ANTIDOTE: British Anti-Lewisite (BAL; dimercaprol) binds to the arsenic in lewisite to decrease the toxicity of this agent. BAL is given by intramuscular (IM) injection as an antidote for whole-body (systemic) health effects of lewis-

ite but has no effect on local lesions of the skin, eyes, or airways. Side effects from BAL are known; administration of BAL should occur only in a hospital setting.

Note: BAL should not be administered to persons with a peanut allergy.

EYE:

Immediately remove the patient/victim from the source of exposure.

Immediately wash eyes with large amounts of tepid water for at least 15 minutes.

If irritatation or pain is severe or persists prolonged eye washing is advised.

Seek medical attention immediately.

INGESTION:

Immediately remove the patient/victim from the source of exposure.

Ensure that the patient/victim has an unobstructed airway.

Do not induce vomiting (emesis).

Administer nothing by mouth (NPO).

Seek medical attention immediately.

INHALATION:

Immediately remove the patient/victim from the source of exposure.

Evaluate respiratory function and pulse.

Ensure that the patient/victim has an unobstructed airway.

If shortness of breath occurs or breathing is difficult (dyspnea), administer oxygen.

Assist ventilation as required. Always use a barrier or bag-valve-mask device.

If breathing has ceased (apnea), provide artificial respiration.

Monitor for and treat spasmodic narrowing of the large airways (bronchospasm), if it occurs.

If evidence of shock or low blood pressure (hypotension) is observed, begin intravenous (IV) fluid administration.

Seek medical attention immediately.

SKIN:

Immediately remove the patient/victim from the source of exposure.

See the decontamination section for patient/victim decontamination procedures.

Skin must be decontaminated within minutes after exposure to limit injury.

Persons exposed to lewisite will seldom be received for medical treatment in time to prevent tissue damage.

Treat any chemical burns with standard burn therapy.

Seek medical attention immediately.

Long-Term Implications

MEDICAL TREATMENT: Fluid and electrolyte balance should be monitored and restored if abnormal. Gastric lavage is contraindicated following ingestion of this agent due to the risk of perforation of the esophagus or upper airway.

DELAYED EFFECTS OF EXPOSURE: There is no substantial evidence to suggest that one exposure to lewisite will cause cancer or that it can cause birth defects in the children of exposed individuals. Chronic respiratory disease may occur following severe airway exposures. Acute, severe injuries to the eye may result in permanently reduced visual acuity or blindness.

EFFECTS OF CHRONIC OR REPEATED EXPOSURE: Information regarding lewisite's potential as a carcinogen, reproductive toxin, or developmental toxin, from chronic or repeated exposure is inconclusive. However, because of lewisite's arsenic content, it is considered a suspect carcinogen. Chronic or repeated exposure to lewisite may lead to arsenical poisoning and an allergic response. Chronic or repeated exposure may cause: in the eyes, conjunctivitis, photophobia, dimness of vision, diplopia, and/or lacrimation; in the nasal and oral cavities, burning sensation, dryness, or breath with a garlic-like odor; in the nervous system, toxic encephalopathy, peripheral neuropathy, or seizures; in the gastrointestinal system, nausea or vomiting; in the respiratory system, obstructive lung disease and bronchitis, although the relationship of this to the arsenical poisoning is unknown; and in the dermatological system, dermatitis, skin ulceration, and basal or squamous cell cancer, the latter only after years of exposure.

ON-SITE FATALITIES

INCIDENT SITE:

Consult with the Incident Commander regarding the agent dispersed, dissemination method, level of PPE required, location, geographic complications (if any), and the approximate number of remains.

Coordinate responsibilities and prepare to enter the scene as part of the evaluation team along with the FBI HazMat Technician, local law enforcement evidence technician, and other relevant personnel.

Begin tracking remains using waterproof tags.

RECOVERY AND ON-SITE MORGUE:

Wear PPE until all remains are deemed free of contamination.

Establish a preliminary (holding) morgue.

Gather evidence, and place it in a clearly labeled impervious container. Hand any evidence over to the FBI.

Remove and tag personal effects.

Perform a thorough external evaluation and a preliminary identification check.

See the Decontamination section for decontamination procedures.

Decontaminate remains before they are removed from the incident site.

ENVIRONMENT/SPILLAGE DISPOSAL: The following methods can be used to decontaminate the environment/spillage disposal:

Do not touch or walk through the spilled agent if at all possible. However, if you must, personnel should wear the appropriate PPE during environmental decontamination. See the PPE section of this card for detailed information.

Keep combustibles (e.g., wood, paper, and oil) away from the spilled agent.

Use water spray to reduce vapors or divert vapor cloud drift. Avoid allowing water runoff to contact the spilled agent.

Do not direct water at the spill or the source of the leak.

Stop the leak if it is possible to do so without risk to personnel, and turn leaking containers so that gas rather than liquid escapes.

Prevent entry into waterways, sewers, basements, or confined areas.

Isolate the area until gas has dispersed.

Ventilate the area.

CHAPTER 6
LUNG DAMAGING AGENTS (CHOKING AGENTS)

PHOSGENE (CG):
Common Names:
Carbonic dichloride
Carbonyl chloride
Chloroformyl chloride
Agent Characteristics

APPEARANCE: Colorless gas above 47 °F (8.2 °C). Fog-like when concentrated. Colorless, fuming liquid below 47 °F (8.2 °C). May have the appearance of a white cloud. Light yellow liquid when refrigerated or compressed.

DESCRIPTION: Phosgene (CG) was originally synthesized in 1812. It was used during WWI by the German army, and has since become part of the chemical arsenal of many countries including the United States. Small amounts of phosgene (CG) exist naturally in the atmosphere from the breakdown of chlorinated compounds. Phosgene is used in the preparation and manufacture of many organic chemicals especially in the dye, pharmaceutical, herbicide, insecticide, metal ore extraction, synthetic foam, resin, polymer, and chlorinating agent industries. Phosgene (CG) may also be released from household paint removers and degreasers when they are used in the presence of heat. Phosgene (CG) is shipped as a liquefied compressed gas in steel cylinders. At low concentrations, phosgene (CG) smells like newly mown hay or green corn. At high concentrations, phosgene (CG) has a strong, suffocating, unpleasant odor. However, the odor is only detectable for a short amount of time when phosgene (CG) is initially released and should not be depended on as a reliable indicator of overexposure.

METHODS OF DISSEMINATION:

Indoor Air: Phosgene (CG) can be released into indoor air as a gas.

Water: Phosgene is unlikely to contaminate water because it breaks down rapidly upon contact with water to produce hydrochloric acid and carbon dioxide.

Food: Phosgene is unlikely to contaminate food because it breaks down rapidly upon contact with water to produce hydrochloric acid and carbon dioxide.

Outdoor Air: Phosgene (CG) can be released into outdoor air as a gas.

Agricultural: If phosgene (CG) is released as a gas, it is highly unlikely to contaminate agricultural products.

ROUTES OF EXPOSURE: Inhalation is the primary route of exposure to phosgene (CG). Ingestion is unlikely, as phosgene (CG) is a gas at room temperature. Exposure to phosgene (CG) may be irritating to the eyes and skin.

Personal Protective Equipment

GENERAL INFORMATION: First Responders should use a NIOSH-certified Chemical, Biological, Radiological, Nuclear (CBRN) Self Contained Breathing Apparatus (SCBA) with a Level A protective suit when entering an

area with an unknown contaminant or when entering an area where the concentration of the contaminant is unknown. Level A protection should be used until monitoring results confirm the contaminant and the concentration of the contaminant.

NOTE: Safe use of protective clothing and equipment requires specific skills developed through training and experience.

LEVEL A: (RED ZONE): Select when the greatest level of skin, respiratory, and eye protection is required. This is the maximum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than the AEGL-2.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A Totally-Encapsulating Chemical Protective (TECP) suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, and a hard hat worn under the TECP suit are optional items.

LEVEL B: (RED ZONE): Select when the highest level of respiratory protection is necessary but a lesser level of skin protection is required. This is the minimum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than AEGL-2. It differs from Level A in that it incorporates a non-encapsulating, splash-protective, chemical-resistant splash suit that provides Level A protection against liquids but is not airtight.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL C: (YELLOW ZONE): Select when the contaminant and concentration of the contaminant are known and the respiratory protection criteria factors for using Air Purifying Respirators (APR) or Powered Air Purifying Respirators (PAPR) are met. This level is appropriate when decontaminating patient/victims.

A NIOSH-certified CBRN tight-fitting APR with a canister-type gas mask or CBRN PAPR for air levels greater than AEGL-2.

A NIOSH-certified CBRN PAPR with a loose-fitting face-piece, hood, or helmet and a filter or a combination organic vapor, acid gas, and particulate cartridge/filter combination or a continuous flow respirator for air levels greater than AEGL-1.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Escape mask, face shield, coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL D: (GREEN ZONE): Select when the contaminant and concentration of the contaminant are known and the concentration is below the appropriate occupational exposure limit or less than AEGL-1 for the stated duration times.

Limited to coveralls or other work clothes, boots, and gloves.

Emergency Response

CHEMICAL DANGERS:

Phosgene (CG) reacts violently with strong oxidants, amines, alkalis, and many metals.

Phosgene (CG) reacts with alcohols and ammonia.

Above 572 °F (300 °C), phosgene (CG) decomposes in the presence of moisture to form hydrochloric acid and carbon dioxide.

In the presence of moisture, phosgene (CG) attacks plastic, rubber, and many metals.

EXPLOSION HAZARDS:

Phosgene is not combustible.

Containers may explode when heated.

Ruptured cylinders may rocket.

FIRE FIGHTING INFORMATION:

Phosgene (CG) is non-combustible.

When heated to decomposition, phosgene (CG) produces toxic and corrosive fumes (hydrogen chloride, carbon monoxide, and chlorine).

For small fires, use dry chemical or carbon dioxide.

For large fires, use water spray, fog, or regular foam. Move containers from the fire area if it is possible to do so without risk to personnel. Do not get water inside containers. Damaged cylinders should be handled only by specialists.

For fire involving tanks, fight the fire from maximum distance or use unmanned hose holders or monitor nozzles. Cool containers with flooding quantities of water until well after the fire is out. Do not direct water at the source of the leak or at safety devices; icing may occur. Withdraw immediately in case of rising sound from venting safety devices or discoloration of tanks. Always stay away from tanks engulfed in fire.

Run-off from fire control may cause pollution.

If the situation allows, control and properly dispose of run-off (effluent).

INITIAL ISOLATION AND PROTECTIVE ACTION DISTANCES:

If a tank, rail car, or tank truck is involved in a fire, isolate it for 1 mi (1600 m) in all directions; also consider initial evacuation for 1 mi (1600 m) in all directions.

Small spills (involving the release of approximately 52.83 gallons (200 liters) or less).

First isolate in all directions 500 ft (150 m).

Then protect persons downwind during the day: 0.8 mi (1.3 km).

Then protect persons downwind during the night: 2.0 mi (3.3 km).

Large spills (involving quantities greater than 52.83 gallons (200 liters))

First isolate in all directions 2500 ft (800 m).

Then protect persons downwind during the day: 4.5 mi (7.3 km).

Then protect persons downwind during the night: 7.0+ mi (11.0+ km).

PHYSICAL DANGERS:

Vapors are heavier than air. They will spread along the ground and collect and stay in poorly-ventilated, low-lying, or confined areas (e.g., sewers, basements, and tanks).

Hazardous concentrations may develop quickly in enclosed, poorly-ventilated, or low-lying areas. Keep out of these areas. Stay upwind.

Phosgene (CG) liquid sinks in water.

NFPA 704 Signal:

Health: 4

Flammability: 0

Reactivity: 1

Special:

Signs/Symptoms

TIME COURSE: Patient/victims exposed to low concentrations of phosgene (CG) vapor may not experience any irritation, or they may have only mild irritation of the upper airways; this allows them to inhale phosgene (CG) for a longer time and more deeply into the lungs. There is a symptom-free (latent) period of 30 minutes to 72 hours, depending on the severity of exposure. The more severe the exposure, the shorter the latency. Physical exertion may bring on shortness of breath or difficulty breathing (dyspnea) or fluid accumulation in the lungs (pulmonary edema). Onset of pulmonary edema within 2 to 6 hours is predictive of severe injury. If the patient/victim survives the initial 48 hours after exposure, recovery is likely.

EFFECTS OF SHORT-TERM (LESS THAN 8-HOURS) EXPOSURE: Phosgene (CG) exerts its toxicity by its action on the body's proteins, as well as through the production of hydrochloric acid. The lung is the major target organ. Irritation of the eyes and upper respiratory tract may be mild, while effects on the cells of the lower airways and lungs may be severe. Heart failure and death may occur as a complication of lung damage.

EYE EXPOSURE:

Gas (high concentrations): Tear production (lacrimation), accumulation of blood (hyperemia), inflammation, and clouding (opacification) of the cornea.

Liquid: Clouding (opacification) of the cornea and delayed perforation.

INGESTION EXPOSURE:

Phosgene (CG) is present as a gas at room temperature, so ingestion is unlikely.

INHALATION EXPOSURE:

Mild: No adverse health effects or only mild upper airway irritation; effects may improve when the patient/victim is removed from exposure; more severe adverse health effects are possible after a delay (latent period).

Mild to moderate: After a symptom-free interval (latent period), irritation of the upper airway, dryness and burning of the throat, painful cough, choking, sense of chest discomfort, difficulty breathing or shortness of breath (dyspnea), spasmodic narrowing of the large airways (bronchospasm), and possible nausea and vomiting (emesis) may occur. Patient/victims with underlying reactive airways or asthma may be at increased risk.

Severe: Rapid accumulation of fluid in the lungs (pulmonary edema); shallow rapid respirations; severe, painful coughing fits producing frothy liquid (sputum); possible upper airway closure (laryngospasm) that may result in sudden death; difficulty breathing or shortness of breath (dyspnea); possible cardiovascular collapse due to low blood oxygen; and low blood pressure secondary to fluid accumulation in the lungs (pulmonary edema).

SKIN EXPOSURE:

Gas: Irritation and redness (erythema) on contact with wet or moist skin.

Severe skin burns or frostbite may occur as a result of contact with compressed liquefied gas.

Decontamination

INTRODUCTION: The purpose of decontamination is to make an individual and/or their equipment safe by physically removing toxic substances quickly and effectively. Care should be taken during decontamination, because absorbed agent can be released from clothing and skin as a gas. Your Incident Commander will provide you with decontaminants specific for the agent released or the agent believed to have been released.

DECONTAMINATION CORRIDOR: The following are recommendations to protect the first responders from the release area:

Position the decontamination corridor upwind and uphill of the hot zone. The warm zone should include two decontamination corridors. One decontamination corridor is used to enter the warm zone and the other for exiting the warm zone into the cold zone. The decontamination zone for exiting should be upwind and uphill from the zone used to enter.

Decontamination area workers should wear appropriate PPE. See the PPE section of this card for detailed information.

A solution of detergent and water (which should have a pH value of at least 8 but should not exceed a pH value of 10.5) should be available for use in decontamination procedures. Soft brushes should be available to remove contamination from the PPE. Labeled, durable 6-mil polyethylene bags should be available for disposal of contaminated PPE.

INDIVIDUAL DECONTAMINATION: The following methods can be used to decontaminate an individual:

Decontamination of First Responder:

Begin washing PPE of the first responder using soap and water solution and a soft brush. Always move in a downward motion (from head to toe). Make sure to get into all areas, especially folds in the clothing. Wash and rinse (using cold or warm water) until the contaminant is thoroughly removed.

Remove PPE by rolling downward (from head to toe) and avoid pulling PPE off over the head. Remove the SCBA after other PPE has been removed.

Place all PPE in labeled durable 6-mil polyethylene bags.

Decontamination of Patient/Victim:

Remove the patient/victim from the contaminated area and into the decontamination corridor.

Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag.

Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds.

Cover the patient/victim to prevent shock and loss of body heat.

Move the patient/victim to an area where emergency medical treatment can be provided.

First Aid.

GENERAL INFORMATION: Initial treatment is primarily supportive. The patient/victim should be kept warm and quiet. Physical exertion should be avoided during treatment and recovery.

ANTIDOTE: There is no antidote for phosgene (CG) toxicity.

EYE:

Immediately remove the patient/victim from the source of exposure.

Immediately wash eyes with large amounts of tepid water for at least 15 minutes.

Eye exposure is unlikely to occur without inhalation exposure.

See Inhalation.

Seek medical attention immediately.

INGESTION:

Not established/determined

INHALATION:

Immediately remove the patient/victim from the source of exposure.

Ensure that the patient/victim has an unobstructed airway.

Evaluate respiratory function and pulse. If shortness of breath, difficulty breathing, or respiratory distress occur, administer oxygen.

Assist ventilation as required. Always use a barrier or bag-valve-mask device.

Monitor the patient/victim for signs of accumulation of fluid in the lungs (pulmonary edema), such as difficulty breathing or shortness of breath (dyspnea) and chest tightness.

Monitor for and treat spasmodic narrowing of airways.

Monitor the patient/victim for signs of whole-body (systemic) effects and administer symptomatic treatment as necessary.

Keep the patient/victim calm and avoid unnecessary exertion or movement.

Seek medical attention immediately.

SKIN:

Immediately remove the patient/victim from the source of exposure.

See the Decontamination section for patient/victim decontamination procedures.

Monitor the patient/victim for signs of whole-body (systemic) effects.

If signs of whole-body (systemic) poisoning appear, see Inhalation for treatment recommendations.

In case of frostbite, protect the injured area from further injury until sustained re-warming can be initiated.

Seek medical attention immediately.

Long-Term Implications

MEDICAL TREATMENT: It has been recommended that patient/victims be monitored for 12 to 48 hours for potential accumulation of fluid in the lungs (pulmonary edema). However, symptoms may be delayed for up to 72 hours. If a patient/victim survives the first 48 hours after exposure, recovery is more likely.

DELAYED EFFECTS OF EXPOSURE: Patient/victims who develop and recover from symptomatic phosgene inhalation are at increased risk for chronic lung damage, increased susceptibility to lung infections, reactive airway dysfunction syndrome (RADS), and an increased risk for sensitivity to airborne irritants.

EFFECTS OF CHRONIC OR REPEATED EXPOSURE: Information is unavailable about the carcinogenicity, developmental toxicity, or reproductive toxicity from chronic or repeated exposure to phosgene (CG). Workers exposed to phosgene (CG) over time (i.e., chronically) experienced an increased rate of adverse health effects and death due to inflammation of the lung and airways, destruction of lung tissue (emphysema), and loss of normal lung function. Chronic low level exposure to phosgene (CG) may cause chronic inflammation and/or infection of the lungs, which may improve with time or may progress to the accumulation of fluid in the lungs (pulmonary edema).

On-Site Fatalities.

INCIDENT SITE:

Consult with the Incident Commander regarding the agent dispersed, dissemination method, level of PPE required, location, geographic complications (if any), and the approximate number of remains.

Coordinate responsibilities and prepare to enter the scene as part of the evaluation team along with the FBI HazMat Technician, local law enforcement evidence technician, and other relevant personnel.

Begin tracking remains using waterproof tags.

RECOVERY AND ON-SITE MORGUE:

Wear PPE until all remains are deemed free of contamination.

Establish a preliminary (holding) morgue.

Gather evidence, and place it in a clearly labeled impervious container. Hand any evidence over to the FBI.

Remove and tag personal effects.

Perform a thorough external evaluation and a preliminary identification check.

See the Decontamination section for decontamination procedures.

Decontaminate remains before they are removed from the incident site.

ENVIRONMENT/SPILLAGE DISPOSAL: The following methods can be used to decontaminate the environment/spillage disposal:

Do not touch or walk through the spilled agent if at all possible. However, if you must, personnel should wear the appropriate PPE during environmental decontamination. See the PPE section of this card for detailed information.

Keep combustibles (e.g., wood, paper, and oil) away from the spilled agent. Use water spray to reduce vapors or divert vapor cloud drift. Avoid allowing water runoff to contact the spilled agent.

Do not direct water at the spill or the source of the leak.

Stop the leak if it is possible to do so without risk to personnel, and turn leaking containers so that gas rather than liquid escapes.

Prevent entry into waterways, sewers, basements, or confined areas.

Isolate the area until gas has dispersed.

CHAPTER 7
BLOOD AGENTS

CYANOGEN CHLORIDE (CK):

Common Names:

Chlorcyan

Chlorine cyanide

Agent Characteristics

APPEARANCE: Colorless, liquid below 55 °F (12.8 °C) or gas above 55 °F (12.8 °C).

DESCRIPTION: Cyanogen chloride (CK) is a highly volatile and toxic chemical asphyxiant that interferes with the body's ability to use oxygen. Exposure to cyanogen chloride (CK) can be rapidly fatal. It has whole-body (systemic) effects, particularly affecting those organ systems most sensitive to low oxy-

gen levels: the central nervous system (brain), the cardiovascular system (heart and blood vessels), and the pulmonary system (lungs). Cyanogen chloride (CK) has strong irritant and choking effects. Its vapors are extremely irritating and corrosive. Cyanogen chloride (CK) is a chemical warfare agent (military designation CK). It is used commercially in chemical synthesis and fumigation.

METHODS OF DISSEMINATION:

Indoor Air: Cyanogen chloride (CK) can be released into indoor air as a liquid spray (aerosol) or as a gas.

Water: Cyanogen chloride (CK) can be used to contaminate water.

Food: Cyanogen chloride (CK) can be used to contaminate food.

Outdoor Air: Cyanogen chloride (CK) can be released into outdoor air as a liquid spray (aerosol) or as a gas.

Agricultural: If cyanogen chloride (CK) is released into the air as a liquid spray (aerosol), it has the potential to contaminate agricultural products. If cyanogen chloride (CK) is released as a gas, it is highly unlikely to contaminate agricultural products.

ROUTES OF EXPOSURE: Cyanogen chloride (CK) can affect the body by inhalation, ingestion, skin contact, or eye contact.

Personal Protective Equipment

GENERAL INFORMATION: First Responders should use a NIOSH-certified Chemical, Biological, Radiological, Nuclear (CBRN) Self Contained Breathing Apparatus (SCBA) with a Level A protective suit when entering an area with an unknown contaminant or when entering an area where the concentration of the contaminant is unknown. Level A protection should be used until monitoring results confirm the contaminant and the concentration of the contaminant.

NOTE: Safe use of protective clothing and equipment requires specific skills developed through training and experience.

LEVEL A: (RED ZONE): Select when the greatest level of skin, respiratory, and eye protection is required. This is the maximum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than the AEGL-2.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A Totally-Encapsulating Chemical Protective (TECP) suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, and a hard hat worn under the TECP suit are optional items.

LEVEL B: (RED ZONE): Select when the highest level of respiratory protection is necessary but a lesser level of skin protection is required. This is

the minimum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than AEGL-2. It differs from Level A in that it incorporates a non-encapsulating, splash-protective, chemical-resistant splash suit that provides Level A protection against liquids but is not airtight.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL C: (YELLOW ZONE): Select when the contaminant and concentration of the contaminant are known and the respiratory protection criteria factors for using Air Purifying Respirators (APR) or Powered Air Purifying Respirators (PAPR) are met. This level is appropriate when decontaminating patient/victims.

A NIOSH-certified CBRN tight-fitting APR with a canister-type gas mask or CBRN PAPR for air levels greater than AEGL-2.

A NIOSH-certified CBRN PAPR with a loose-fitting face-piece, hood, or helmet and a filter or a combination organic vapor, acid gas, and particulate cartridge/filter combination or a continuous flow respirator for air levels greater than AEGL-1.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Escape mask, face shield, coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL D: (GREEN ZONE): Select when the contaminant and concentration of the contaminant are known and the concentration is below the appropriate occupational exposure limit or less than AEGL-1 for the stated duration times.

Limited to coveralls or other work clothes, boots, and gloves.

Emergency Response

CHEMICAL DANGERS:

Cyanogen chloride (CK) decomposes on heating, producing toxic and corrosive fumes (hydrogen cyanide, hydrochloric acid, and nitrogen oxides). See the emergency response cards for hydrogen cyanide and hydrochloric acid.

Cyanogen chloride (CK) reacts slowly with water or water vapor to form toxic hydrogen cyanide and hydrogen chloride. See the emergency response cards for hydrogen cyanide and hydrogen chloride.

Cyanogen chloride (CK) is incompatible with or may react with most basic and acidic solvents including water.

Cyanogen chloride (CK) is unstable; it may be stabilized (inhibited) to prevent polymerization.

EXPLOSION HAZARDS:

Upper and lower explosive (flammable) limits in air are not available for cyanogen chloride (CK).

Containers may explode when heated or if they are contaminated with water.

Ruptured cylinders may rocket.

FIRE FIGHTING INFORMATION:

Fire will produce irritating, corrosive, and/or toxic gases.

Cyanogen chloride (CK) may burn, but it does not ignite readily.

For small fires, use dry chemical or carbon dioxide.

For large fires, use water spray, fog, or regular foam. Move containers from the fire area if it is possible to do so without risk to personnel. Do not get water inside containers. Damaged cylinders should be handled only by specialists.

For fire involving tanks, fight the fire from maximum distance or use unmanned hose holders or monitor nozzles. Cool containers with flooding quantities of water until well after the fire is out. Do not direct water at the source of the leak or at safety devices; icing may occur. Withdraw immediately in case of rising sound from venting safety devices or discoloration of tanks. Always stay away from tanks engulfed in fire.

Run-off from fire control may cause pollution.

If the situation allows, control and properly dispose of run-off (effluent).

INITIAL ISOLATION AND PROTECTIVE ACTION DISTANCES:

If a tank, rail car, or tank truck is involved in a fire, isolate it for 1.0 mi (1600 m) in all directions; also consider initial evacuation for 1.0 mi (1600 m) in all directions.

Small spills (involving the release of approximately 52.83 gallons (200 liters) or less)

First isolate in all directions: 200 ft (60 m).

Then protect persons downwind during the day: 0.4 mi (0.6 km).

Then protect persons downwind during the night: 1.8 mi (2.8 km).

Large spills (involving quantities greater than 52.83 gallons (200 liters)):

First isolate in all directions: 1400 ft (450 m).

Then protect persons downwind during the day: 2.7 mi (4.3 km).

Then protect persons downwind during the night: 6.3 mi (10.1 km).

Small spills (involving the release of approximately 52.83 gallons (200 liters) or less), when cyanogen chloride is used as a weapon :

First isolate in all directions: 200 ft (60 m).

Then protect persons downwind during the day: 0.4 mi (0.7 km).

Then protect persons downwind during the night: 1.5 mi (2.5 km).

Large spills (involving quantities greater than 52.83 gallons (200 liters)), when cyanogen chloride is used as a weapon:

First isolate in all directions: 1300 ft (420 m).

Then protect persons downwind during the day: 2.5 mi (4.1 km).

Then protect persons downwind during the night: 5.0 mi (8.1 km).

PHYSICAL DANGERS:

Vapors may be heavier than air. They will spread along the ground and collect and stay in poorly-ventilated, low-lying, or confined areas (e.g., sewers, basements, and tanks).

Hazardous concentrations may develop quickly in enclosed, poorly-ventilated, or low-lying areas. Keep out of these areas. Stay upwind.

NFPA 704 Signal:

Health: 4

Flammability: 0

Reactivity: 2

Special:

Signs/Symptoms

TIME COURSE: Early symptoms of cyanide poisoning include lightheadedness, giddiness, rapid breathing, nausea, vomiting (emesis), feeling of neck constriction and suffocation, confusion, restlessness, and anxiety. Accumulation of fluid in the lungs (pulmonary edema) may complicate severe intoxications. Rapid breathing is soon followed by respiratory depression/respiratory arrest (cessation of breathing). Severe cyanide poisonings progress to stupor, coma, muscle spasms (in which head, neck, and spine are arched backwards), convulsions (seizures), fixed and dilated pupils, and death. The CNS is the most sensitive target organ of cyanide poisoning. Cardiovascular effects require higher cyanide doses than those necessary for CNS effects. In serious poisonings, the skin is cold, clammy, and diaphoretic. Blue discoloration of the skin may be a late finding. Severe signs of oxygen deprivation in the absence of blue discoloration of the skin suggest cyanide poisoning.

EFFECTS OF SHORT-TERM (LESS THAN 8-HOURS) EXPOSURE: Cyanogen chloride (CK) can be rapidly fatal. It severely irritates the eyes, skin, and respiratory tract. It produces whole-body (systemic) effects by interfering with oxygen utilization at the cellular level, with profound central nervous system (CNS), cardiovascular, and respiratory effects. Severe signs of decreased oxygen supply to the tissues (hypoxia) in the absence of bluish discoloration of the skin (cyanosis) are characteristic; cyanosis usually occurs late in the course of poisoning, at the stage of circulatory collapse and cessation of breathing (apnea).

EYE EXPOSURE:

Intense irritation, severe spasmodic blinking (blepharospasm), and tear production (lacrimation).

Contact with only the eyes has not been known to result in whole-body (systemic) toxicity, although this is a possibility.

See Inhalation Exposure.

INGESTION EXPOSURE:

Possible bitter, acrid burning taste, followed by constriction or numbness of the throat, salivation, nausea, and vomiting (emesis).

Whole-body (systemic) toxicity can occur.

See Inhalation Exposure.

INHALATION EXPOSURE:

Mild to moderate: CNS effects: headache, confusion, anxiety, dizziness, weakness (malaise), and loss of consciousness. Cardiovascular effects: palpitations. Respiratory effects: respiratory tract irritation, difficulty breathing or shortness of breath (dyspnea), and transient increase in the rate and depth of breathing (hyperpnea). GI effects: nausea and vomiting (emesis).

Severe: CNS effects: coma, seizures, and dilated pupils (mydriasis). Cardiovascular effects: shock, abnormal or disordered heart rhythms (dysrhythmias), critically low blood pressure, and cardiac arrest. Respiratory effects: abnormally rapid, followed by abnormally slow respirations; accumulation of fluid in the lungs (pulmonary edema); and respiratory arrest. Eye effects: dilated pupils, inflammation of the surface of the eye, and temporary blindness.

SKIN EXPOSURE:

Irritation.

Contact with gas or liquefied gas may cause burns, severe injury, and/or frostbite.

Contact with the skin can contribute to whole-body (systemic) toxicity.

See Inhalation Exposure.

Decontamination

INTRODUCTION: The purpose of decontamination is to make an individual and/or their equipment safe by physically removing toxic substances quickly and effectively. Care should be taken during decontamination, because absorbed agent can be released from clothing and skin as a gas. Your Incident Commander will provide you with decontaminants specific for the agent released or the agent believed to have been released.

DECONTAMINATION CORRIDOR: The following are recommendations to protect the first responders from the release area:

Position the decontamination corridor upwind and uphill of the hot zone. The warm zone should include two decontamination corridors. One decontamination corridor is used to enter the warm zone and the other for exiting the warm zone into the cold zone. The decontamination zone for exiting should be upwind and uphill from the zone used to enter.

Decontamination area workers should wear appropriate PPE. See the PPE section of this card for detailed information.

A solution of detergent and water (which should have a pH value of at least 8 but should not exceed a pH value of 10.5) should be available for use in decontamination procedures. Soft brushes should be available to remove contamination from the PPE. Labeled, durable 6-mil polyethylene bags should be available for disposal of contaminated PPE.

INDIVIDUAL DECONTAMINATION: The following methods can be used to decontaminate an individual:

Decontamination of First Responder:

Begin washing PPE of the first responder using soap and water solution and a soft brush. Always move in a downward motion (from head to toe). Make sure to get into all areas, especially folds in the clothing. Wash and rinse (using cold or warm water) until the contaminant is thoroughly removed.

Remove PPE by rolling downward (from head to toe) and avoid pulling PPE off over the head. Remove the SCBA after other PPE has been removed.

Place all PPE in labeled durable 6-mil polyethylene bags.

Decontamination of Patient/Victim:

Remove the patient/victim from the contaminated area and into the decontamination corridor.

Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag.

Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds.

Cover the patient/victim to prevent shock and loss of body heat.

Move the patient/victim to an area where emergency medical treatment can be provided.

First Aid

GENERAL INFORMATION: Careful observation, supplemental oxygen, and supportive care may be sufficient therapy for the patient/victim who does not exhibit physical findings of cyanide toxicity. For the patient/victim exhibiting physical findings of cyanide toxicity, initial treatment consists of administration of antidotes under a physician's direction, respiratory and circulatory support (oxygen and IV fluids), correction of chemical imbalances in the blood, and seizure control. Speed is critical. Avoid mouth-to-mouth resuscitation regardless of route of exposure. Avoid contact with vomitus, which may off-gas hydrogen cyanide.

ANTIDOTE: Amyl nitrite, sodium nitrite, and sodium thiosulfate are antidotes for cyanide toxicity; however, amyl nitrite and sodium nitrite should not be administered to patient/victims suffering from smoke inhalation. In these cases, only administer sodium thiosulfate. The described administration of nitrites is based on a patient having normal hemoglobin levels. Below normal hemoglobin levels require titration of nitrites.

For mild to moderate physical findings such as nausea, vomiting, palpitations, confusion, anxiety, dizziness (vertigo), and/or abnormally fast or deep respiration (hyperventilation):

Child (less than 55 lb (25 kg)) Observe the patient/victim and administer 0.75 mL per pound of a 25 % solution (1.65 mL per kilogram of a 25 % solution) of sodium thiosulfate intravenously over a period of 10 minutes.

Adult Observe the patient/victim and administer 12.5 g of a 25 % solution (50 mL of a 25 % solution) of sodium thiosulfate intravenously over a period of 10 minutes.

For severe physical findings such as coma; cessation of breathing (apnea); seizures; slowness of the heart rate, usually to fewer than 60 beats per minute (bradycardia); abnormally low blood pressure (hypotension); bluish skin coloring due to abnormally low levels of oxygen in the blood (cyanosis); irregular heart beat (dysrhythmias); and/or accumulation of fluid in the lungs (pulmonary edema):

Child (less than 55 lb (25 kg)) Until sodium nitrite becomes available, break one ampule of amyl nitrite into a cloth. Out of every minute, hold the cloth containing amyl nitrite in front of the patient's mouth for 30 seconds, and then remove it for 30 seconds, until sodium nitrite can be administered. A new ampule of amyl nitrite should be broken into a cloth every 3 minutes. Discontinue use of amyl nitrite when sodium nitrite becomes available. Administration of an entire dose (10 mL of a 3 % solution) of sodium nitrite to a child can produce overwhelming lethal methemoglobinemia. Therefore, children should receive 0.15 mL per pound of body weight of sodium nitrite (0.33 mL per kg body weight of 3% sodium nitrite) over a period of 5 to 20 minutes.

Next, administer 0.75 mL per pound body weight of 25 % sodium thiosulfate (1.65 mL per kilogram body weight of 25 % sodium thiosulfate) intravenously over a period of 10 minutes. If physical findings persist for 30 minutes after antidote administration, sodium nitrite and sodium thiosulfate may be re-administered at half their original respective doses. However, methemoglobin levels should be monitored and not allowed to exceed 40 %.

If a child weighs more than 55 lb (25 kg), administer antidote as described for the adult (see below).

Adult Until sodium nitrite becomes available, break one ampule of amyl nitrite into a cloth. Out of every minute, hold the cloth containing amyl nitrite in front of the patient's mouth for 30 seconds, and then remove it for 30 seconds, until sodium nitrite can be administered. A new ampule of amyl nitrite should be broken into a cloth every 3 minutes. Discontinue use of amyl nitrite when sodium nitrite becomes available. Administer 300 mg of a 3 % solution (10 mL of a 3 % solution) of sodium nitrite intravenously over a period of 5 to 20 minutes.

Next, administer 12.5 g (50 mL of a 25 % solution) of sodium thiosulfate intravenously over a period of 10 minutes. If physical findings persist for 30 minutes after antidote administration, sodium nitrite and sodium thiosulfate

may be readministered at half their original respective doses. However, methemoglobin levels should be monitored and not allowed to exceed 40%.

EYE:

Immediately remove the patient/victim from the source of exposure.

Immediately wash eyes with large amounts of tepid water for at least 15 minutes.

Monitor the patient/victim for signs of whole-body (systemic) effects.

If signs of whole-body (systemic) poisoning appear, see the Inhalation section for treatment recommendations.

Seek medical attention immediately.

INGESTION:

Immediately remove the patient/victim from the source of exposure.

Establish secure large-bore IV access.

Ensure that the patient/victim has an unobstructed airway.

Do not induce vomiting (emesis).

Immediately administer 100 % oxygen.

Prepare a cyanide antidote kit, for use under a physician's direction, for symptomatic patient/victims. See the Antidote section for antidote administration procedures.

Treat seizures with benzodiazepines.

Seek medical attention immediately.

INHALATION:

Immediately remove the patient/victim from the source of exposure.

Evaluate respiratory function and pulse.

Ensure that the patient/victim has an unobstructed airway.

Immediately administer 100 % oxygen.

Assist ventilation as required.

If breathing has ceased (apnea), provide artificial respiration.

Establish secure large-bore intravenous (IV) access.

Prepare a cyanide antidote kit, for use under a physician's direction, for symptomatic patient/victims. See the Antidote section for antidote administration procedures.

Monitor for respiratory distress and accumulation of fluid in the lungs (pulmonary edema).

Seek medical attention immediately.

SKIN:

Immediately remove the patient/victim from the source of exposure.

See the Decontamination section for patient/victim decontamination.

Monitor the patient/victim for signs of whole-body (systemic) effects.

If signs of whole-body (systemic) poisoning appear, see the Inhalation First Aid section for treatment recommendations.

Seek medical attention immediately.

Long-Term Implications.

MEDICAL TREATMENT: Evidence for the benefit of gastric decontamination in cases of cyanide ingestion is limited at best and should come after all other known life-saving measures have been instituted. Gastric lavage (stomach pumping) is recommended only if it can be done shortly after ingestion (generally within 1 hour), in an emergency department, and after the airway has been secured. Activated charcoal may be administered as a slurry (240 mL water/30 g charcoal). Usual dose: 25 to 100 g in adults/adolescents, 25 to 50 g in children (1 to 12 years), and 1 g/kg in infants less than 1 year old. Patient/victims who have ingested cyanogen chloride (CK) or patient/victims who have direct skin or eye contact should be observed in the Emergency Department for at least 4 to 6 hours for the development of delayed symptoms. Patient/victims with significant inhalation exposure should be monitored for the accumulation of fluid in the lungs (pulmonary edema), which occurs more rapidly with exposure to cyanogen chloride (CK) than with exposure to other cyanides.

DELAYED EFFECTS OF EXPOSURE: Usually death occurs rapidly or there is prompt recovery. Survivors of severe cyanide exposures may suffer brain damage due to a direct effect of the poison (toxin) on nerve cells, or to a lack of oxygen, or possibly due to insufficient blood circulation. Examples of long-term neurological effects caused by cyanide poisoning include personality changes, memory loss, and disturbances in movement (both voluntary and involuntary movement disorders); some damage may be permanent.

EFFECTS OF CHRONIC OR REPEATED EXPOSURE: Information is unavailable about the carcinogenicity, developmental toxicity, or reproductive toxicity from chronic or repeated exposure to cyanogen chloride (CK). Effects of chronic or repeated exposure to cyanogen chloride (CK) are similar to those of cyanide and other cyanide compounds. Chronically exposed workers may complain of headache, eye irritation, easy fatigue, chest discomfort, palpitations, loss of appetite (anorexia), and nosebleeds (epistaxis). Exposure to small amounts of cyanide compounds over long periods of time is reported to cause loss of appetite, headache, weakness, nausea, dizziness, and symptoms of irritation of the upper respiratory tract and eyes.

On-Site Fatalities.

INCIDENT SITE:

Consult with the Incident Commander regarding the agent dispersed, dissemination method, level of PPE required, location, geographic complications (if any), and the approximate number of remains.

Coordinate responsibilities and prepare to enter the scene as part of the evaluation team along with the FBI HazMat Technician, local law enforcement evidence technician, and other relevant personnel.

Begin tracking remains using waterproof tags.

RECOVERY AND ON-SITE MORGUE:

Wear PPE until all remains are deemed free of contamination.

Establish a preliminary (holding) morgue.

Gather evidence, and place it in a clearly labeled impervious container.
Remove and tag personal effects.
Perform a thorough external evaluation and a preliminary identification check.
See the Decontamination section for decontamination procedures.
Decontaminate remains before they are removed from the incident site.

HYDROGEN CYANIDE (AC)

Common Names:

Formonitrile
Hydrocyanic acid
Prussic acid
Agent Characteristics

APPEARANCE: Colorless or pale blue liquid below 78°F (25.6°C), colorless gas above 78 °F (25.6 °C).

DESCRIPTION: Hydrogen cyanide (AC) is a systemic chemical asphyxiant. It interferes with the normal use of oxygen by nearly every organ of the body. Exposure to hydrogen cyanide (AC) can be rapidly fatal. It has whole-body (systemic) effects, particularly affecting those organ systems most sensitive to low oxygen levels: the central nervous system (brain), the cardiovascular system (heart and blood vessels), and the pulmonary system (lungs). Hydrogen cyanide (AC) is a chemical warfare agent (military designation, AC). It is used commercially for fumigation, electroplating, mining, chemical synthesis, and the production of synthetic fibers, plastics, dyes, and pesticides. Hydrogen cyanide (AC) gas has a distinctive bitter almond odor (others describe a musty «old sneakers smell»), but a large proportion of people cannot detect it; the odor does not provide adequate warning of hazardous concentrations. It also has a bitter burning taste and is often used as a solution in water.

METHODS OF DISSEMINATION:

Indoor Air: Hydrogen cyanide (AC) can be released into indoor air as a liquid spray (aerosol) or as a gas.

Water: Hydrogen cyanide (AC) can be used to contaminate water.

Food: Hydrogen cyanide (AC) can be used to contaminate food.

Outdoor Air: Hydrogen cyanide (AC) can be released into outdoor air as a liquid spray (aerosol) or as a gas.

Agricultural: If hydrogen cyanide (AC) is released into the air as a liquid spray (aerosol), it has the potential to contaminate agricultural products. If hydrogen cyanide (AC) is released as a gas, it is highly unlikely to contaminate agricultural products.

ROUTES OF EXPOSURE: Hydrogen cyanide (AC) can affect the body by ingestion, inhalation, skin contact, or eye contact.

Personal Protective Equipment.

GENERAL INFORMATION: First Responders should use a NIOSH-certified Chemical, Biological, Radiological, Nuclear (CBRN) Self Contained

Breathing Apparatus (SCBA) with a Level A protective suit when entering an area with an unknown contaminant or when entering an area where the concentration of the contaminant is unknown. Level A protection should be used until monitoring results confirm the contaminant and the concentration of the contaminant.

NOTE: Safe use of protective clothing and equipment requires specific skills developed through training and experience.

LEVEL A: (RED ZONE): Select when the greatest level of skin, respiratory, and eye protection is required. This is the maximum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than the AEGL-2.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A Totally-Encapsulating Chemical Protective (TECP) suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, and a hard hat worn under the TECP suit are optional items.

LEVEL B: (RED ZONE): Select when the highest level of respiratory protection is necessary but a lesser level of skin protection is required. This is the minimum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than AEGL-2. It differs from Level A in that it incorporates a non-encapsulating, splash-protective, chemical-resistant splash suit that provides Level A protection against liquids but is not airtight.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL C: (YELLOW ZONE): Select when the contaminant and concentration of the contaminant are known and the respiratory protection criteria factors for using Air Purifying Respirators (APR) or Powered Air Purifying Respirators (PAPR) are met. This level is appropriate when decontaminating patient/victims.

A NIOSH-certified CBRN tight-fitting APR with a canister-type gas mask or CBRN PAPR for air levels greater than AEGL-2.

A NIOSH-certified CBRN PAPR with a loose-fitting face-piece, hood, or helmet and a filter or a combination organic vapor, acid gas, and particulate cartridge/filter combination or a continuous flow respirator for air levels greater than AEGL-1.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Escape mask, face shield, coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL D: (GREEN ZONE): Select when the contaminant and concentration of the contaminant are known and the concentration is below the appropriate occupational exposure limit or less than AEGL-1 for the stated duration times.

Limited to coveralls or other work clothes, boots, and gloves.

Emergency Response

CHEMICAL DANGERS:

Hydrogen cyanide (AC) is unstable with heat, alkaline materials, and water.

Hydrogen cyanide (AC) reacts with amines, oxidants, acids, sodium hydroxide, calcium hydroxide, sodium carbonate, caustic substances, and ammonia.

Hydrogen cyanide (AC) may polymerize at 122 °F to 140 °F (50 °C to 60 °C); polymerization can occur violently in the presence of heat, alkaline materials, or moisture.

EXPLOSION HAZARDS:

Hydrogen cyanide (AC) gas mixes well with air, and explosive mixtures are easily formed.

Confined polymerization can cause container failure and a violent explosion.

Hydrogen cyanide (AC) can decompose explosively on contact with alkaline materials.

Explosive potential is severe when hydrogen cyanide (AC) is exposed to heat or flame or to alkaline agents.

Lower explosive (flammable) limit in air (LEL), 5.6 %; upper explosive (flammable) limit in air (UEL), 40 %.

The agent or its vapors present a vapor explosion and poison (toxic) hazard indoors, outdoors, or in sewers.

Run-off to sewers may create an explosion hazard.

Containers may explode when heated.

Ruptured cylinders may rocket.

INITIAL ISOLATION AND PROTECTIVE ACTION DISTANCES:

If a tank, rail car, or tank truck is involved in a fire, isolate it for 1 mi (1600 m) in all directions; also consider initial evacuation for 1 mi (1600 m) in all directions.

Small spills (when used as a weapon):

First isolate in all directions: 200 ft (60 m).

Then protect persons downwind during the day: 0.1 mi (0.2 km).

Then protect persons downwind during the night: 0.3 mi (0.5 km).

Large spills (when used as a weapon):
First isolate in all directions: 1500 ft (500 m).
Then protect persons downwind during the day: 1.0 mi (1.7 km).
Then protect persons downwind during the night: 2.4 mi (3.9 km).
Small spills:
First isolate in all directions: 100 ft (30 m).
Then protect persons downwind during the day: 0.1 mi (0.1 km).
Then protect persons downwind during the night: 0.3 mi (0.4 km).
Large spills:
First isolate in all directions: 500 ft (150 m).
Then protect persons downwind during the day: 0.8 mi (1.3 km).
Then protect persons downwind during the night: 2.3 mi (3.7 km).

PHYSICAL DANGERS:

Hazardous concentrations may develop quickly in enclosed or poorly-ventilated areas.

Hydrogen cyanide (AC) gas mixes well with air; explosive mixtures are easily formed.

NFPA 704 Signal:

Health: 4
Flammability: 4
Reactivity: 2
Special:

Signs/Symptoms

TIME COURSE: Effects occur extremely rapidly following exposure to hydrogen cyanide (AC). After inhalation exposure, symptoms begin within seconds to minutes; death may occur within minutes. After skin exposure, onset of symptoms may be immediate or delayed for 30 to 60 minutes. Ingestion of hydrogen cyanide (AC) solutions or cyanide salts can be rapidly fatal. The time of onset of effects depends on the concentration and duration of exposure.

EFFECTS OF SHORT-TERM (LESS THAN 8-HOURS) EXPOSURE: Early symptoms of cyanide poisoning include lightheadedness, giddiness, rapid breathing, nausea, vomiting (emesis), feeling of neck constriction and suffocation, confusion, restlessness, and anxiety. Accumulation of fluid in the lungs (pulmonary edema) may complicate severe intoxications. Rapid breathing is soon followed by respiratory depression/respiratory arrest (cessation of breathing). Severe cyanide poisonings progress to stupor, coma, muscle spasms (in which head, neck, and spine are arched backwards), convulsions (seizures),

fixed and dilated pupils, and death. The CNS is the most sensitive target organ of cyanide poisoning. Cardiovascular effects require higher cyanide doses than those necessary for CNS effects. In serious poisonings, the skin is cold, clammy, and diaphoretic. Blue discoloration of the skin may be a late finding. Severe signs of oxygen deprivation in the absence of blue discoloration of the skin suggest cyanide poisoning.

EYE EXPOSURE:

Irritation.

Contact with only the eyes does not normally result in whole-body (systemic) toxicity.

Contact with the eyes can contribute to whole-body (systemic) toxicity. See Inhalation Exposure.

INGESTION EXPOSURE:

Burning sensation in mouth and throat, nausea, vomiting (emesis), and abdominal pain.

Whole-body (systemic) toxicity can occur. See Inhalation Exposure.

INHALATION EXPOSURE:

Mild to moderate: CNS effects: headache, confusion, anxiety, dizziness, weakness (malaise), and loss of consciousness. Cardiovascular effects: palpitations. Respiratory effects: respiratory tract irritation, difficulty breathing or shortness of breath (dyspnea), and transient increase in the rate and depth of breathing (hyperpnea). GI effects: nausea and vomiting (emesis).

Severe: CNS effects: coma, seizures, and dilated pupils (mydriasis). Cardiovascular effects: shock, abnormal or disordered heart rhythms (dysrhythmias), critically low blood pressure, and cardiac arrest. Respiratory effects: abnormally rapid, followed by abnormally slow respirations; accumulation of fluid in the lungs (pulmonary edema); and respiratory arrest. Eye effects: dilated pupils, inflammation of the surface of the eye, and temporary blindness.

SKIN EXPOSURE:

Irritation.

Absorption through the skin is rapid and can contribute to whole-body (systemic) toxicity. See Inhalation Exposure.

Absorption through the skin occurs more readily when ambient temperature and relative humidity are high.

Decontamination

INTRODUCTION: The purpose of decontamination is to make an individual and/or their equipment safe by physically removing toxic substances quickly and effectively. Care should be taken during decontamination, because absorbed agent can be released from clothing and skin as a gas. Your Incident Commander will provide you with decontaminants specific for the agent released or the agent believed to have been released.

DECONTAMINATION CORRIDOR: The following are recommendations to protect the first responders from the release area:

Position the decontamination corridor upwind and uphill of the hot zone. The warm zone should include two decontamination corridors. One decontamination corridor is used to enter the warm zone and the other for exiting the warm zone into the cold zone. The decontamination zone for exiting should be upwind and uphill from the zone used to enter.

Decontamination area workers should wear appropriate PPE. See the PPE section of this card for detailed information.

A solution of detergent and water (which should have a pH value of at least 8 but should not exceed a pH value of 10.5) should be available for use in decontamination procedures. Soft brushes should be available to remove contamination from the PPE. Labeled, durable 6-mil polyethylene bags should be available for disposal of contaminated PPE.

INDIVIDUAL DECONTAMINATION: The following methods can be used to decontaminate an individual:

Decontamination of First Responder:

Begin washing PPE of the first responder using soap and water solution and a soft brush. Always move in a downward motion (from head to toe). Make sure to get into all areas, especially folds in the clothing. Wash and rinse (using cold or warm water) until the contaminant is thoroughly removed.

Remove PPE by rolling downward (from head to toe) and avoid pulling PPE off over the head. Remove the SCBA after other PPE has been removed.

Place all PPE in labeled durable 6-mil polyethylene bags.

Decontamination of Patient/Victim:

Remove the patient/victim from the contaminated area and into the decontamination corridor.

Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag.

Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds.

Cover the patient/victim to prevent shock and loss of body heat.

Move the patient/victim to an area where emergency medical treatment can be provided.

First Aid

GENERAL INFORMATION: Careful observation, supplemental oxygen, and supportive care may be sufficient therapy for the patient/victim who does not exhibit physical findings of cyanide toxicity. For the patient/victim exhibiting physical findings of cyanide toxicity, initial treatment consists of administration of antidotes under a physician's direction, respiratory and circulatory support (oxygen and IV fluids), correction of chemical imbalances in the blood, and seizure control. Speed is critical. Avoid mouth-to-mouth resuscitation regardless of route of exposure. Avoid contact with vomitus, which may off-gas hydrogen cyanide.

ANTIDOTE: Amyl nitrite, sodium nitrite, and sodium thiosulfate are antidotes for cyanide toxicity; however, amyl nitrite and sodium nitrite should not be administered to patient/victims suffering from smoke inhalation. In these cases, only administer sodium thiosulfate. The described administration of nitrites is based on a patient having normal hemoglobin levels. Below normal hemoglobin levels require titration of nitrites.

For mild to moderate physical findings such as nausea, vomiting, palpitations, confusion, anxiety, dizziness (vertigo), and/or abnormally fast or deep respiration (hyperventilation):

Child (less than 55 lb (25 kg)) Observe the patient/victim and administer 0.75 mL per pound of a 25 % solution (1.65 mL per kilogram of a 25 % solution) of sodium thiosulfate intravenously over a period of 10 minutes.

Adult Observe the patient/victim and administer 12.5 g of a 25 % solution (50 mL of a 25 % solution) of sodium thiosulfate intravenously over a period of 10 minutes.

For severe physical findings such as coma; cessation of breathing (apnea); seizures; slowness of the heart rate, usually to fewer than 60 beats per minute (bradycardia); abnormally low blood pressure (hypotension); bluish skin coloring due to abnormally low levels of oxygen in the blood (cyanosis); irregular heart beat (dysrhythmias); and/or accumulation of fluid in the lungs (pulmonary edema):

Child (less than 55 lb (25 kg)) Until sodium nitrite becomes available, break one ampule of amyl nitrite into a cloth. Out of every minute, hold the cloth containing amyl nitrite in front of the patient's mouth for 30 seconds, and then remove it for 30 seconds, until sodium nitrite can be administered. A new ampule of amyl nitrite should be broken into a cloth every 3 minutes. Discontinue use of amyl nitrite when sodium nitrite becomes available. Administration of an entire dose (10 mL of a 3% solution) of sodium nitrite to a child can produce overwhelming lethal methemoglobinemia. Therefore, children should receive 0.15 mL per pound of body weight of sodium nitrite (0.33 mL per kg body weight of 3 % sodium nitrite) over a period of 5 to 20 minutes.

Next, administer 0.75 mL per pound body weight of 25 % sodium thiosulfate (1.65 mL per kilogram body weight of 25 % sodium thiosulfate) intravenously over a period of 10 minutes. If physical findings persist for 30 minutes after antidote administration, sodium nitrite and sodium thiosulfate may be readministered at half their original respective doses. However, methemoglobin levels should be monitored and not allowed to exceed 40 %.

If a child weighs more than 55 lb (25 kg), administer antidote as described for the adult (see below).

Adult Until sodium nitrite becomes available, break one ampule of amyl nitrite into a cloth. Out of every minute, hold the cloth containing amyl nitrite in front of the patient's mouth for 30 seconds, and then remove it for 30 seconds, until sodium nitrite can be administered. A new ampule of amyl nitrite should be broken into a cloth every 3 minutes. Discontinue use of amyl nitrite when sodi-

um nitrite becomes available. Administer 300 mg of a 3 % solution (10 mL of a 3 % solution) of sodium nitrite intravenously over a period of 5 to 20 minutes.

Next, administer 12.5 g (50 mL of a 25 % solution) of sodium thiosulfate intravenously over a period of 10 minutes. If physical findings persist for 30 minutes after antidote administration, sodium nitrite and sodium thiosulfate may be readministered at half their original respective doses. However, methemoglobin levels should be monitored and not allowed to exceed 40 %.

EYE:

Immediately remove the patient/victim from the source of exposure.

Immediately wash eyes with large amounts of tepid water for at least 15 minutes.

Monitor the patient/victim for signs of whole-body (systemic) effects.

If signs of whole-body (systemic) poisoning appear, see the Inhalation section for treatment recommendations.

Seek medical attention immediately.

INGESTION:

Immediately remove the patient/victim from the source of exposure.

Establish secure large-bore IV access.

Ensure that the patient/victim has an unobstructed airway.

Do not induce vomiting (emesis).

Immediately administer 100% oxygen.

Prepare a cyanide antidote kit, for use under a physician's direction, for symptomatic patient/victims. See the Antidote section for antidote administration procedures.

Treat seizures with benzodiazepines.

Seek medical attention immediately.

INHALATION:

Immediately remove the patient/victim from the source of exposure.

Evaluate respiratory function and pulse.

Ensure that the patient/victim has an unobstructed airway.

Immediately administer 100 % oxygen.

Assist ventilation as required.

If breathing has ceased (apnea), provide artificial respiration.

Establish secure large-bore intravenous (IV) access.

Prepare a cyanide antidote kit, for use under a physician's direction, for symptomatic patient/victims. See the Antidote section for antidote administration procedures.

Monitor for respiratory distress.

Seek medical attention immediately.

SKIN:

Immediately remove the patient/victim from the source of exposure.

See the Decontamination section for patient/victim decontamination procedures.

Monitor the patient/victim for signs of whole-body (systemic) effects.

If signs of whole-body (systemic) poisoning appear, see the Inhalation section for treatment recommendations.

Seek medical attention immediately.

Long-Term Implications

MEDICAL TREATMENT: Evidence for the benefit of gastric decontamination in cases of cyanide ingestion is limited at best and should come after all other known life-saving measures have been instituted. Gastric lavage (stomach pumping) is recommended only if it can be done shortly after ingestion (generally within 1 hour), in an emergency department, and after the airway has been secured. Activated charcoal may be administered as a slurry (240 mL water/30 g charcoal). Usual dose: 25 to 100 g in adults/adolescents, 25 to 50 g in children (1 to 12 years), and 1 g/kg in infants less than 1 year old. Patient/victims who have ingested hydrogen cyanide (AC) solutions or patient/victims who have direct skin or eye contact should be observed in the Emergency Department for at least 4 to 6 hours for the development of delayed symptoms. Patient/victims with significant inhalation exposure should be monitored for the accumulation of fluid in the lungs (pulmonary edema), which may occur up to 24 to 72 hours following exposure.

DELAYED EFFECTS OF EXPOSURE: Usually death occurs rapidly or there is prompt recovery. Survivors of severe cyanide exposures may suffer brain damage due to a direct effect of the poison (toxin) on nerve cells, or to a lack of oxygen, or possibly due to insufficient blood circulation. Examples of long-term neurological effects caused by cyanide poisoning include personality changes, memory loss, and disturbances in movement (both voluntary and involuntary movement disorders); some damage may be permanent.

EFFECTS OF CHRONIC OR REPEATED EXPOSURE: Hydrogen cyanide (AC) has not been classified for cancer-causing (carcinogenic) effects, and no carcinogenic effects have been reported for hydrogen cyanide (AC). No reproductive or developmental effects of hydrogen cyanide (AC) have been reported in humans. Chronically exposed workers may complain of headache, eye irritation, easy fatigue, chest discomfort, palpitations, loss of appetite (anorexia), and nosebleeds (epistaxis). Workers such as electroplaters and picklers, who are daily exposed to cyanide solutions, may develop a "cyanide" rash, characterized by itching and by macular, papular, and vesicular eruptions. Exposure to small amounts of cyanide compounds over long periods of time is reported to cause loss of appetite, headache, weakness, nausea, dizziness, and symptoms of irritation of the upper respiratory tract and eyes.

On-Site Fatalities.

INCIDENT SITE:

Consult with the Incident Commander regarding the agent dispersed, dissemination method, level of PPE required, location, geographic complications (if any), and the approximate number of remains.

Coordinate responsibilities and prepare to enter the scene as part of the evaluation team along with the FBI HazMat Technician, local law enforcement evidence technician, and other relevant personnel.

Begin tracking remains using waterproof tags.

RECOVERY AND ON-SITE MORGUE:

Wear PPE until all remains are deemed free of contamination.

Establish a preliminary (holding) morgue.

Gather evidence, and place it in a clearly labeled impervious container. Hand any evidence over to the FBI.

Remove and tag personal effects.

Perform a thorough external evaluation and a preliminary identification check.

See the Decontamination section for decontamination procedures.

Decontaminate remains before they are removed from the incident site.

ENVIRONMENT/SPILLAGE DISPOSAL: The following methods can be used to decontaminate the environment/spillage disposal:

Do not touch or walk through the spilled agent if at all possible. However, if you must, personnel should wear the appropriate PPE during environmental decontamination. See the PPE section of this card for detailed information.

Keep combustibles (e.g., wood, paper, and oil) away from the spilled agent. Use water spray to reduce vapors or divert vapor cloud drift. Avoid allowing water runoff to contact the spilled agent.

Do not direct water at the spill or the source of the leak.

Stop the leak if it is possible to do so without risk to personnel, and turn leaking containers so that gas rather than liquid escapes.

Prevent entry into waterways, sewers, basements, or confined areas.

Isolate the area until gas has dispersed.

ARSINE

Description

Arsine is a colorless, flammable, and highly toxic gas. It has a garlic-like or fishy odor that can be detected at concentrations of 0.5 ppm and above. Because arsine is nonirritating and produces no immediate symptoms, persons exposed to hazardous levels may be unaware of its presence. Arsine is water soluble. It is generally shipped in cylinders as a liquefied compressed gas (NIOSH 2005). Exposure may occur when arsine gas is generated while metals or crude ores containing arsenic impurities are treated with acid (HSDB 2007).

Routes of Exposure

Inhalation

Inhalation is the major route of exposure. The odor threshold of arsine is 10-fold greater than the Occupational Safety and Health Administration (OSHA)

permissible exposure limit. **Odor is not an adequate indicator of arsine presence and does not provide reliable warning of hazardous concentrations**. Arsine is heavier than air and hazardous concentrations may develop quickly in enclosed, poorly ventilated, or low-lying areas. Initial symptoms (malaise, dizziness, nausea, abdominal pain, and dyspnea) may develop within several hours of exposure to 3 ppm of arsine (AIHA 1999).

Children exposed to the same levels of arsine as adults may receive larger doses because they have relatively greater lung surface area:body weight ratios and higher minute volumes:weight ratios. In addition, they may be exposed to higher levels than adults in the same location because of their short stature and the higher levels of arsine found nearer to the ground.

Skin/Eye Contact

There is little information about direct toxic effects of arsine on the skin or eyes, or about absorption through the skin (AIHA 1999). Exposure to liquefied arsine (the compressed gas) can result in frostbite (NIOSH 2005).

Ingestion

Ingestion of arsine itself is unlikely because it is a gas at room temperature. However, metal arsenides are solids that can react with acidic gastric contents, releasing arsine gas in the stomach.

Sources/Uses

Arsine gas is formed when arsenic-containing materials react with freshly formed hydrogen in water or acids (NIOSH 2005). Exposure may result when arsenic containing metals (i.e., metal vats) undergo acid washes. Unintentional exposures have also occurred during refining of ores (e.g., lead, copper, zinc, iron, and antimony ores) that contain arsenic. Arsine is used as a doping agent in the semiconductor industry and in the manufacture of crystals for fiberoptics and computer chips. It is used infrequently in galvanizing, soldering, etching, burnishing, and lead plating (HSDB 2007; IPCS 1997).

Standards and Guidelines

OSHA PEL (permissible exposure limit) = 0.05 ppm (averaged over an 8-hour workshift) (OSHA 2006).

NIOSH IDLH (immediately dangerous to life or health) = 3 ppm (NIOSH 2005). NIOSH considers arsine to be a potential occupational carcinogen.

EPA AEGL-2 (Acute Exposure Guideline Level-2) for arsine = 0.3 ppm (10-minute) to 0.02 ppm (8-hour); AEGL 1 not recommended due to steep dose-response relationship, mechanism of toxicity, and because toxicity occurs at or below the odor threshold (EPA 2007).

Physical Properties

Description: Colorless, nonirritating gas at room temperature.

Warning properties: Inadequate; garlic-1ike or fishy smell; odor threshold of 0.5 ppm (IPCS 1997)

Molecular weight: 77.9 daltons (HSDB 2007)

Boiling point (760 mm Hg): -80.5 °F (-62.5 °C) (HSDB 2007)

Vapor pressure: 11,000 mm Hg at 68 °F (20 °C) (HSDB 2007)

Gas density: 2.7 (air = 1) (NIOSH 2005)

Water solubility: 28 mg/100 mL at 68 °F (20 °C) (HSDB 2007)

Flammability: Extremely flammable; may be ignited by heat, sparks, or flames. Vapors may travel to a source of ignition and flash back (HSDB 2007).

Incompatibilities

Arsine reacts with strong oxidizers, chlorine, and nitric acid. Arsine decomposes above 446 °F (230 °C) (NIOSH 2007).

Health Effects

Arsine is a highly toxic gas and may be fatal if inhaled in sufficient quantities. Its primary toxic effect is due to hemolysis resulting in renal failure.

Common initial symptoms of exposure include malaise, headache, thirst, shivering, abdominal pain, and dyspnea. These symptoms usually occur within 30-60 minutes with heavy exposure, but can be delayed for 2-24 hours. Absence of early symptoms does not necessarily indicate a nontoxic exposure.

Hemoglobinuria usually occurs within hours, jaundice within 1 or 2 days.

Acute Exposure

After absorption by the lungs, arsine enters red blood cells (RBC) where different processes may contribute to hemolysis and impairment of oxygen transport. Inhibition of catalase may lead to accumulation of hydrogen peroxide which, as an oxidizer, destroys red cell membranes and may contribute to arsine-induced conversion of Fe^{+2} to Fe^{+3}, which also impairs oxygen transport. Arsine preferentially binds to hemoglobin, and is oxidized to an arsenic dihydride intermediate and elemental arsenic, both of which are hemolytic agents. Arsine toxicity involves depletion of reduced glutathione. Therefore, people deficient in the enzyme glucose-6-phosphate-dehydrogenase (G6PD) are more susceptible to hemolysis following arsine exposure. Pre-existing cardiopulmonary or renal conditions, iron deficiency, and/or pre-existing anemia may result in more severe outcomes if hemolysis occurs (AIHA 1999; Beliles 1994; Woods 1995).

Contact with the skin or eyes is not expected to result in systemic toxicity. Ingestion of arsine is unlikely, but ingestion of metallic arsenides can lead to arsine gas production and toxicity.

Hematologic

Acute intravascular hemolysis develops within hours and may be severe during the first 2 or 3 days following exposure. Free hemoglobin levels in plasma rise (levels greater than 2 g/dL have been reported). Anemia ensues subsequent to hemolysis. Anemia may develop quickly and be severe. Leukocytosis and signs of intravascular coagulation can be observed during the hemolytic phase (IPCS 1997). Methemoglobinemia can be of concern in infants and toddlers. Children may be more vulnerable to loss of effectiveness of hemoglobin because of their relative anemia compared to adults.

Respiratory

A garlic odor may be present on the breath. Delayed accumulation of fluid in the lungs may occur after massive exposure. Dyspnea may be due to lack of oxygen secondary to hemolysis (HSDB 2007; IPCS 1997).

Children may be more vulnerable to gas exposure because of relatively higher minute ventilation per kg and failure to recognize the need to promptly evacuate an area when exposed.

Renal

Kidney failure due to acute tubular necrosis is a significant sequela of arsine exposure. Hemoglobin in the urine is thought to be the major cause of damage to the kidneys; however, a direct toxic effect of arsine or deposition of the arsine-hemoglobin-haptoglobin complex may also play a role. Urinalysis shows large amounts of protein and free hemoglobin usually without intact RBCs. Urine may be unusually colored (e.g., brown, red, orange, or greenish). Decreased urinary output may develop within 24-48 hours (HSDB 2007; IPCS 1997).

Gastrointestinal

Nausea, vomiting, and crampy abdominal pain are among the first signs of arsine poisoning. Onset varies from a few minutes to 24 hours after exposure (HSDB 2007).

Dermal

The characteristic bronze tint of the skin caused by arsine toxicity is induced by hemolysis and may be caused by hemoglobin deposits. The bronze coloration is not jaundice, although jaundice may develop later as a result of significant hemolysis (HSDB 2007).

Contact with liquefied arsine (compressed gas) can cause frostbite (HSDB 2007).

CNS

Headache is often an early sign of exposure. CNS disorders can develop several days after severe exposure; signs include restlessness, memory loss, diso-

rientation, and agitation. Some exposed persons experience signs of peripheral nerve damage 1–2 weeks after exposure. There are case reports of polyneuropathy developing 1-6 months after arsine exposure (AIHA 1999; Reigart and Roberts 1999).

Hepatic

Right upper quadrant pain, hepatomegaly, elevated serum globulin, elevated liver enzymes and prolonged prothrombin time have been observed (AIHA 1999).

Musculoskeletal

Skeletal muscle injury or necrosis have been reported (HSDB 2007). Muscle pain and twitches, myoglobinuria, elevated levels of serum creatine phosphokinase (CPK), and aldolase have been observed.

Cardiovascular

Cardiovascular effects may include moderate and transient sinus tachycardia secondary to hemolysis or anemia, hypovolemia or acute pulmonary edema, hypotension and cardiovascular shock due to direct effects on the myocardium and hyperkalemia, elevation of the T-wave (ECG) and various degrees of heart block, and general vasoconstriction due to peripheral hypoxia (IPCS 1997).

Ophthalmic/Ocular

Watery eyes, photophobia, blurred vision, and red staining of the conjunctiva may appear early after exposure (IPCS 1997).

Chronic Exposure

Chronic arsine exposure can result in gastrointestinal upset, anemia, and damage to lungs, kidneys, liver, nervous system, heart, and blood-forming organs (HSDB 2007; IPCS 1997). There is little information regarding health effects of chronic low-level exposures to arsine.

Carcinogenicity

There are no data on the carcinogenicity of arsine in humans or in experimental animals. However, arsine is oxidized to the same trivalent and pentavalent forms of arsenic as those seen after drinking-water or inhalation exposure to arsenic compounds known to present a cancer hazard. The Department of Health and Human Services (DHHS), the International Agency for Research on Cancer (IARC), and the Environmental Protection Agency (EPA) have classified inorganic arsenic as a human carcinogen based on sufficient evidence from human data.

Reproductive and Developmental Effects

Arsine should be treated as a potential teratogenic agent. Although the reproductive effects of acute or chronic exposure to arsine are unknown, some re-

lated inorganic arsenicals produce a broad spectrum of adverse developmental effects in animals. Animal studies indicated that in arsine-exposed mothers, arsenic crosses the placenta and reaches the fetus (AIHA 1999); however, no adverse developmental effects were observed.

Prehospital Management

Although small amounts of arsine gas can be trapped in the victimâ€™s clothing or hair after an overwhelming exposure, these quantities are not likely to create a hazard for response personnel outside the Hot Zone.

The odor of arsine is not always detected during serious exposures. Since symptoms may be delayed, ALL exposure victims should be evaluated at a medical facility.

Toxic effects may be delayed for up to 2-24 hours after exposure.

There is no specific antidote for arsine. Treatment is symptomatic and consists of measures to support respiratory, vascular, and renal function.

Hot Zone

Rescuers should be trained and appropriately attired before entering the Hot Zone. If the proper equipment is not available, or if rescuers have not been trained in its use, assistance should be obtained from a local or regional HAZMAT team or other properly equipped response organization.

Rescuer Protection

Arsine is a highly toxic systemic poison.

Respiratory Protection: Positive-pressure, self-contained breathing apparatus (SCBA) is recommended in response situations that involve exposure to potentially unsafe levels of arsine (NIOSH 2005).

Skin Protection: Chemical-protective clothing is not generally required because arsine gas is not absorbed through the skin and does not cause skin irritation. However, contact with the liquid (compressed gas) can cause frostbite injury to the skin or eyes (NIOSH 2005).

ABC Reminders

Quickly establish a patent airway, ensure adequate respiration and pulse. If trauma is suspected, maintain cervical immobilization manually and apply a cervical collar and a backboard when feasible. Apply direct pressure to stop any heavy bleeding.

Victim Removal

If victims can walk, lead them out of the Hot Zone to the Decontamination Zone. Victims who are unable to walk may be removed on backboards or gurneys; if these are not available, carefully carry or drag victims to safety.

Victims with chemically-induced acute disorders may suffer from anxiety, especially children who may be separated from a parent or other adult.

Decontamination Zone

Victims who have exposure only to arsine gas do not need decontamination. They may be transferred immediately to the Support Zone. In rare cases of liquefied arsine-contamination, remove and double-bag contaminated clothing.

In rare cases of liquefied arsine dermal contamination, immediately flush exposed areas with running water for at least 15 minutes (IPCS 1997).

Use caution to avoid hypothermia when decontaminating victims, particularly children or the elderly. Use blankets or warmers after decontamination as needed.

Support Zone

Support Zone personnel require no specialized protective gear if the victim has been exposed only to arsine gas.

ABC Reminders

Quickly establish a patent airway, ensure adequate respiration and pulse. Stabilize the cervical spine with a collar and a backboard if trauma is suspected. Administer supplemental oxygen as required. Assist ventilation with a bag-valve-mask device if necessary. Apply direct pressure to control bleeding. Evaluate for respiratory tract irritation, bronchitis, or pneumonitis (HSDB 2007).

In cases of dermal and ocular exposure, immediately flush exposed areas with running water for at least 15 minutes (IPCS 1997).

Use caution to avoid hypothermia when decontaminating victims, particularly children or the elderly. Use blankets or warmers after decontamination as needed.

Advanced Treatment

In cases of respiratory compromise, secure airway and support respiration according to advanced life support (ALS) protocols.

If evidence of shock or hypotension is observed, begin fluid administration. For adults with systolic pressure less than 80 mm Hg, bolus perfusion of 1,000 mL/hour intravenous saline or lactated Ringerâ€™s solution may be appropriate. Higher adult systolic pressures may necessitate lower perfusion rates. For children with compromised perfusion, administer a 20 mL/kg bolus of normal saline over 10–20 minutes, followed by reassessment and further management as clinically appropriate. Monitor fluid balance and avoid fluid overload if renal failure supervenes (Reigart and Roberts 1999); monitor plasma electrolytes to detect disturbances (particularly hyperkalemia) as early as possible. Monitor hematocrit.

Because of possible severe hemolysis, ensure adequate oxygenation by arterial blood gas measurement or pulse oxygenation monitoring. Ensure adequate hydration by starting intravenous fluids. The use of diuretics such as furosemide

to maintain urinary flow is an important consideration and should be performed under medical base control.

Transport to Medical Facility

Report to the base station and the receiving medical facility the condition of the patient, treatment given, and estimated time of arrival at the medical facility.

Multi-Casualty Triage

Consult with the base station physician or the regional poison control center for advice regarding triage of multiple victims.

It is difficult to determine at the scene which persons have had the most serious exposures and are likely to develop severe hemolysis; therefore, all persons who have potential exposure should be transported to a medical facility for evaluation.

Persons who have smelled a garlic- or fish-like odor should be transported first.

Emergency Department Management

Although small amounts of arsine gas can be trapped in the victimâ€™s clothing or hair after an overwhelming exposure, these quantities are not likely to create a hazard for hospital personnel away from the scene.

Arsine poisoning causes acute intravascular hemolysis, which may lead to renal failure. Arsine gas does not produce acute arsenic intoxication.

Even if arsineâ€™s odor was not detected at the scene, those present could have been seriously exposed. All exposure victims should be evaluated and observed.

There is no specific antidote for arsine. Treatment consists of measures to support vascular, renal, hematologic, and respiratory function.

Critical Care Area

Patients exposed only to arsine gas do not need decontamination.

ABC Reminders

Evaluate and support airway, breathing, and circulation according to ALS protocols. Establish intravenous access in symptomatic patients. Monitor cardiac rhythm.

Monitor fluid balance carefully to avoid fluid overload if renal failure supervenes (Reigart and Roberts 1999); monitor plasma electrolytes to detect disturbances (particularly hyperkalemia) as early as possible, and monitor hematocrit.

Patients who are comatose or hypotensive should be treated in the conventional manner.

Consider dopamine for hypotension or oligonuria, or norepinephrine in cases of severe resistant shock.

Observe patients who have inhaled arsine for up to 24 hours. Follow up as clinically indicated.

Inhalation Exposure

Administer supplemental oxygen by mask to patients who have respiratory symptoms. Treat patients who have bronchospasm with aerosolized bronchodi-

lators. The use of bronchial sensitizing agents in situations of multiple chemical exposures may pose additional risks. Also consider the health of the myocardium before choosing which type of bronchodilator should be administered. Cardiac sensitizing agents may be appropriate; however, the use of cardiac sensitizing agents after exposure to certain chemicals may pose enhanced risk of cardiac arrhythmias (especially in the elderly). Arsine poisoning is not known to pose additional risk during the use of bronchial or cardiac sensitizing agents.

Consider racemic epinephrine aerosol for children who develop stridor. Dose 0.25–0.75 mL of 2.25 % racemic epinephrine solution; repeat every 20 minutes as needed while observing for myocardial variability.

If hemolysis develops, initiate urinary alkalinization. Add 50–100 mEq of sodium bicarbonate to one liter of 5 % dextrose in 0.25 normal saline and administer intravenously at a rate that maintains urine output at 2 -3 mL/kg/hour. Maintain alkaline urine (i.e., pH >7.5) until urine is hemoglobin free. Closely monitor serum electrolytes, calcium, BUN, creatinine, hemoglobin, and hematocrit (Ellenhorn 1997).

Consider hemodialysis if renal failure is severe. (Although hemodialysis will assist the patient who has renal failure, it will not effectively remove the arsine-hemoglobin or arsine-haptoglobin complexes deposited in the renal tubules.) Blood transfusions may be necessary if hemolysis causes severe anemia (Reigart and Roberts 1999).

Skin Exposure

In case of frostbite injury, irrigate with lukewarm (42 °C) water according to standard treatment.

Eye Exposure

In case of frostbite injury, ensure that thorough warming with lukewarm water or saline has been completed. Examine the eyes for corneal damage and treat appropriately. Immediately consult an ophthalmologist for patients who have corneal injuries.

Antidotes and Other Treatments

There are no antidotes for arsine poisoning.

Do not administer arsenic chelating drugs. Although BAL (British Anti-Lewisite, dimercaprol) and other chelating agents are acceptable for arsenic poisoning, they are not effective antidotes for arsine poisoning and are not recommended (HSDB 2007).

Laboratory Tests

If significant exposure is a possibility and transfusion is considered, obtain a blood sample for type and screen. Laboratory tests to determine hemolysis in-

clude CBC with peripheral smear, urinalysis, and plasma free hemoglobin and haptoglobin analyses. These tests should be performed in all cases of suspected poisoning. Other useful studies include renal-function tests (e.g., BUN, creatinine), and determinations of serum electrolytes and bilirubin levels. Urinary arsenic may be elevated for a few weeks following arsine exposure and may provide some index of the extent of exposure.

Disposition and Follow-up

Decisions to admit or discharge a patient should be based on exposure history, physical examination, and test results.

Delayed Effects

All patients who have suspected arsine exposure should be carefully observed for 24 hours, including hourly urine output. Onset of hemolysis may be delayed for up to 24 hours, and acute renal failure may not become evident for as long as 72 hours after exposure.

Patient Release

Patients who have no signs of hemolysis may be discharged after 24 hours of observation with instructions to seek medical care promptly if symptoms develop (see the *Arsine-Patient Information Sheet* below). Released patients should also be instructed to rest and to drink plenty of fluids.

Follow-up

Obtain the name of the patientâ€™s primary care physician so that the hospital can send a copy of the ED visit to the patientâ€™s doctor.

All patients should have repeat urine and blood laboratory tests in 12–24 hours. Patients who have corneal injuries should be reexamined within 24 hours.

If severe hemolysis has occurred, anemia may persist for several weeks (AIHA1999).

Polyneuropathy and alteration in mental status are reported to have followed arsine poisoning after a latency of 1-6 months (Reigart and Roberts 1999). Depending on the severity of arsine exposure, patients should be evaluated periodically by their physician for several months; these examinations should include hematological and urinalysis tests.

Reporting

If a work-related incident has occurred, you may be legally required to file a report; contact your state or local health department.

Other persons may still be at risk in the setting where this incident occurred. If the incident occurred in the workplace, discussing it with company personnel may prevent future incidents. If a public health risk exists, notify your

state or local health department or other responsible public agency. When appropriate, inform patients that they may request an evaluation of their workplace from OSHA or NIOSH. See Appendix III for a list of agencies that may be of assistance.

CARBON MONOXIDE

Carbon monoxide (CO) is an odorless, colorless, poisonous gas that can cause sudden illness and death if present in sufficient concentration in the ambient air. When power outages occur during emergencies such as hurricanes or winter storms, the use of alternative sources of fuel or electricity for heating, cooling, or cooking can cause CO to build up in a home, garage, or camper and poison the people and animals inside. Generators, grills, camp stoves, or other gasoline, propane, natural gas, or charcoal-burning devices should never be used inside a home, basement, garage, or camper - or even outside near an open window or window air conditioner.

How to Recognize CO Poisoning: The symptoms and signs of carbon monoxide poisoning are variable and nonspecific. The most common symptoms of CO poisoning are headache, dizziness, weakness, nausea, vomiting, chest pain, and altered mental status.

The clinical presentation of CO poisoning is the result of its underlying systemic toxicity. Its effects are caused not only by impaired oxygen delivery but also by disrupting oxygen utilization and respiration at the cellular level, particularly in high-oxygen demand organs (i.e., heart and brain).

Symptoms of severe CO poisoning include malaise, shortness of breath, headache, nausea, chest pain, irritability, ataxia, altered mental status, other neurologic symptoms, loss of consciousness, coma, and death; signs include tachycardia, tachypnea, hypotension, various neurologic findings including impaired memory, cognitive and sensory disturbances; metabolic acidosis, arrhythmias, myocardial ischemia or infarction, and noncardiogenic pulmonary edema, although any organ system might be involved.

With a focused history, exposure to a CO source may become apparent. Appropriate and prompt diagnostic testing and treatment is very important.

Red Flags: No fever associated with symptoms, history of exposure, multiple patients with similar complaints.

Sources of CO Poisoning.

Gas-powered generators.

Charcoal grills, propane stoves, and charcoal briquettes for both cooking and heating indoors.

Motor vehicles.

Fire.

Boats.

Power washers and other gas powered tools.

At-risk Populations include:

Babies and infants.

The elderly.

People with chronic heart disease, anemia or respiratory illness.

Evaluation.

Diagnosis is based on a suggestive history and physical findings coupled with confirmatory testing. Patients should be examined for other conditions, including smoke inhalation, trauma, medical illness, or intoxication.

Neurological exam should include an assessment of cognitive function such as a Mini-Mental Status Exam

All women of childbearing age who are suspected of having CO poisoning should have a pregnancy test.

Confirmation of diagnosis

The key to confirming the diagnosis is measuring the patient's carboxyhemoglobin (COHb) level.

Carbon Monoxide levels can be tested either in whole blood or exhaled air.

It is important to know how much time has elapsed since the patient has left the toxic environment, because that will impact the COHb level. If the patient has been breathing normal room air for several hours, COHb testing may be less useful.

The most common technology available in hospital laboratories for analyzing the blood is the multiple wavelength spectrophotometer, also known as a CO-oximeter. Venous or arterial blood may be used for testing.

A fingertip pulse CO-oximeter can be used to measure heart rate and oxygen saturation, and COHb levels. The conventional two-wavelength pulse oximeter is not accurate when COHb is present.

An elevated COHb level of 2 % for non-smokers and >9 % COHb level for smokers strongly supports a diagnosis of CO poisoning.

COHb levels do not correlate well with severity of illness, outcomes or response to therapy so it is important to assess clinical symptoms and history of exposure when determining type and intensity of treatment.

Other testing, such as a fingerstick blood sugar, alcohol and toxicology screen, head CT scan or lumbar puncture may be needed to exclude other causes of altered mental status when the diagnosis of carbon monoxide poisoning is inconclusive.

Note: carbon monoxide can be produced endogenously as a byproduct of heme metabolism. Patients with sickle cell disease can have an elevated COHb level as a result of hemolytic anemia or hemolysis.

Guidance for Management of Confirmed or Suspected CO Poisoning

Administer 100 % oxygen until the patient is symptom-free, usually about 4–5 hours. Serial neurologic exams should be performed to assess progress, and to detect the signs of developing cerebral edema.

Consider hyperbaric oxygen therapy (HBO) therapy when the patient has a COHb level of more than 25–30 %, there is evidence of cardiac involvement,

severe acidosis, transient or prolonged unconsciousness, neurological impairment, abnormal neuropsychiatric testing, or the patient is ≥ 36 years in age. HBO is also administered at lower COHb (< 25 %) levels if suggested by clinical condition and/history of exposure.

Hyperbaric oxygenis the treatment of choice for pregnant women, even if they are less severely poisoned. Hyperbaric oxygen is safe to administer and international consensus favors it as part of a more aggressive role in treating pregnant women.

Other Considerations

Cardiac injury during poisoning increases risk of mortality over 10 years following poisoning, so in patients with severe CO poisoning, it may be important to perform an EKG and measurement of troponin and cardiac enzymes.

Chest radiography is recommended for seriously poisoned patients, especially those with loss of consciousness or cardiopulmonary signs and symptoms. Brain computed tomography or MRI is also recommended in these cases; these tests may show signs of cerebral infarction secondary to hypoxia or ischemia.

All discharged patients should be warned of possible delayed neurological complications and given instructions on what to do if these occur. Follow-up should include a repeat medical and neurological exam in 2 weeks.

CHAPTER 8
TOXIC ALCOHOLS

METHANOL

Common Names:

Carbinol
Methyl alcohol
Wood alcohol

Agent Characteristics

APPEARANCE: Colorless watery liquid.

DESCRIPTION: Methanol is a toxic alcohol that is used industrially as a solvent, pesticide, and alternative fuel source. It also occurs naturally in humans, animals, and plants. Foods such as fresh fruits and vegetables, fruit juices, fermented beverages, and diet soft drinks containing aspartame are the primary sources of methanol in the human body. Most methanol poisonings occur as a result of drinking beverages contaminated with methanol or from drinking methanol-containing products. In the industrial setting, inhalation of high concentrations of methanol vapor and absorption of methanol through the skin are as effective as the oral route in producing toxic effects. The characteristic pungent (alcohol) odor of methanol does not provide sufficient warning of low levels of exposure.

METHODS OF DISSEMINATION:

Indoor Air: Methanol can be released into indoor air as a liquid spray (aerosol).

Water: Methanol can be used to contaminate water.

Food: Methanol may be used to contaminate food.

Outdoor Air: Methanol can be released into outdoor air as a liquid spray (aerosol).

Agricultural: If methanol is released into the air as a liquid spray (aerosol), it has the potential to contaminate agricultural products.

ROUTES OF EXPOSURE: Methanol can be absorbed into the body by inhalation, ingestion, skin contact, or eye contact. Ingestion is an important route of exposure.

Personal Protective Equipment

GENERAL INFORMATION: First Responders should use a NIOSH-certified Chemical, Biological, Radiological, Nuclear (CBRN) Self Contained Breathing Apparatus (SCBA) with a Level A protective suit when entering an area with an unknown contaminant or when entering an area where the concentration of the contaminant is unknown. Level A protection should be used until monitoring results confirm the contaminant and the concentration of the contaminant.

NOTE: Safe use of protective clothing and equipment requires specific skills developed through training and experience.

LEVEL A: (RED ZONE): Select when the greatest level of skin, respiratory, and eye protection is required. This is the maximum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than the AEGL-2.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A Totally-Encapsulating Chemical Protective (TECP) suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, and a hard hat worn under the TECP suit are optional items.

LEVEL B: (RED ZONE): Select when the highest level of respiratory protection is necessary but a lesser level of skin protection is required. This is the minimum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than AEGL-2. It differs from Level A in that it incorporates a non-encapsulating, splash-protective, chemical-resistant splash suit that provides Level A protection against liquids but is not airtight.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL C: (YELLOW ZONE): Select when the contaminant and concentration of the contaminant are known and the respiratory protection criteria factors for using Air Purifying Respirators (APR) or Powered Air Purifying Respirators (PAPR) are met. This level is appropriate when decontaminating patient/victims.

A NIOSH-certified CBRN tight-fitting APR with a canister-type gas mask or CBRN PAPR for air levels greater than AEGL-2.

A NIOSH-certified CBRN PAPR with a loose-fitting face-piece, hood, or helmet and a filter or a combination organic vapor, acid gas, and particulate cartridge/filter combination or a continuous flow respirator for air levels greater than AEGL-1.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Escape mask, face shield, coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL D: (GREEN ZONE): Select when the contaminant and concentration of the contaminant are known and the concentration is below the appropriate occupational exposure limit or less than AEGL-1 for the stated duration times.

Limited to coveralls or other work clothes, boots, and gloves.

Emergency Response

CHEMICAL DANGERS:

Methanol reacts violently with strong oxidants, causing a fire and explosion hazard.

EXPLOSION HAZARDS:

Mixtures of methanol vapor and air are explosive.

Lower explosive (flammable) limit in air (LEL), 6.0 %; upper explosive (flammable) limit in air (UEL), 36 %.

Agent presents a vapor explosion and poison (toxic) hazard indoors, outdoors, or in sewers.

Run-off to sewers may create an explosion hazard.

Containers may explode when heated.

FIRE FIGHTING INFORMATION:

Methanol is highly flammable.

The agent will be easily ignited by heat, sparks, or flames.

Fire will produce irritating, corrosive, and/or toxic gases.

Vapors may travel to the source of ignition and flash back.

Run-off to sewers may create a fire hazard.

Caution: The agent has a very low flash point. Use of water spray when fighting fires may be inefficient.

For small fires, use dry chemical, carbon dioxide, water spray, or alcohol-resistant foam.

For large fires, use water spray, fog, or alcohol-resistant foam. Move containers from the fire area if it is possible to do so without risk to personnel. Dike fire control water for later disposal; do not scatter the agent. Use water spray or fog; do not use straight streams.

For fire involving tanks or car/trailer loads, fight the fire from maximum distance or use unmanned hose holders or monitor nozzles. Cool containers with flooding quantities of water until well after the fire is out. Withdraw immediately in case of rising sound from venting safety devices or discoloration of tanks. Always stay away from tanks engulfed in fire.

For massive fire, use unmanned hose holders or monitor nozzles; if this is impossible, withdraw from the area and let the fire burn.

Run-off from fire control or dilution water may cause pollution.

If the situation allows, control and properly dispose of run-off (effluent).

INITIAL ISOLATION AND PROTECTIVE ACTION DISTANCES:

If a tank, rail car, or tank truck is involved in a fire, isolate it for 0.5 mi (800 m) in all directions; also consider initial evacuation for 0.5 mi (800 m) in all directions.

This agent is not included in the DOT ERG 2004 Table of Initial Isolation and Protective Action Distances.

In the DOT ERG 2004 orange-bordered section of the guidebook, there are public safety recommendations to isolate a methanol (Guide 131) spill or leak area immediately for at least 150 ft (50 m) in all directions.

PHYSICAL DANGERS:

Methanol vapors may be heavier than air. They will spread along the ground and collect and stay in poorly-ventilated, low-lying, or confined areas (e.g., sewers, basements, and tanks).

Hazardous concentrations may develop quickly in enclosed, poorly-ventilated, or low-lying areas. Keep out of these areas. Stay upwind.

Liquid agent is lighter than water.

NFPA 704 Signal:

Health: 1

Flammability: 3

Reactivity: 0

Special:

Signs/Symptoms

TIME COURSE: Adverse health effects from methanol poisoning may not become apparent until after an asymptomatic period of 1 to 72 hours.

EFFECTS OF SHORT-TERM (LESS THAN 8-HOURS) EXPOSURE: Methanol's toxicity is due to its metabolic products. The by-products of methanol metabolism cause an accumulation of acid in the blood (metabolic acidosis), blindness, and death. Initial adverse health effects due to methanol poisoning include drowsiness, a reduced level of consciousness (CNS depression), confusion, headache, dizziness, and the inability to coordinate muscle movement (ataxia). Other adverse health effects may include nausea, vomiting (emesis), and heart and respiratory (cardiopulmonary) failure. Prognosis is poor in patient/victims with coma or seizure and severe metabolic acidosis (pH <7). Early on after methanol exposure, there may be a relative absence of adverse health effects. This does not imply insignificant toxicity. Methanol toxicity worsens as the degree of metabolic acidosis increases, and thus, becomes more severe as the time between exposure and treatment increases.

EYE EXPOSURE:

Irritation, redness, and pain.

INGESTION EXPOSURE:

Ingestion of methanol may cause a wide range of adverse health effects:

Neurological: headache, dizziness, agitation, acute mania, amnesia, decreased level of consciousness including coma, and seizure.

Gastrointestinal: Nausea, vomiting, lack of an appetite (anorexia), severe abdominal pain, gastrointestinal bleeding (hemorrhage), diarrhea, liver function abnormalities, and inflammation of the pancreas (pancreatitis).

Ophthalmologic: visual disturbances, blurred vision, sensitivity to light (photophobia), visual hallucinations (misty vision, skin over the eyes, snowstorm, dancing spots, flashes), partial to total loss of vision, and rarely eye pain. Visual examination may reveal abnormal findings. Fixed dilated pupils are a sign of severe exposure to methanol.

Other: Electrolyte imbalances. Kidney failure, blood in the urine (hematuria), and muscle death at the cellular level (rhabdomyolysis) have been reported in severe poisonings. Fatal cases often present with fast heart rate (tachycardia) or slow heart rate (bradycardia) and an increased rate of respiration. Low blood pressure (hypotension) and respiratory arrest occur when death is imminent.

INHALATION EXPOSURE:

See Ingestion Exposure.

SKIN EXPOSURE:

Irritation.

See Ingestion Exposure.

Decontamination

INTRODUCTION: The purpose of decontamination is to make an individual and/or their equipment safe by physically removing toxic substances quickly and effectively. Care should be taken during decontamination, because

absorbed agent can be released from clothing and skin as a gas. Your Incident Commander will provide you with decontaminants specific for the agent released or the agent believed to have been released.

DECONTAMINATION CORRIDOR: The following are recommendations to protect the first responders from the release area:

Position the decontamination corridor upwind and uphill of the hot zone. The warm zone should include two decontamination corridors. One decontamination corridor is used to enter the warm zone and the other for exiting the warm zone into the cold zone. The decontamination zone for exiting should be upwind and uphill from the zone used to enter.

Decontamination area workers should wear appropriate PPE. See the PPE section of this card for detailed information.

A solution of detergent and water (which should have a pH value of at least 8 but should not exceed a pH value of 10.5) should be available for use in decontamination procedures. Soft brushes should be available to remove contamination from the PPE. Labeled, durable 6-mil polyethylene bags should be available for disposal of contaminated PPE.

INDIVIDUAL DECONTAMINATION: The following methods can be used to decontaminate an individual:

Decontamination of First Responder:

Begin washing PPE of the first responder using soap and water solution and a soft brush. Always move in a downward motion (from head to toe). Make sure to get into all areas, especially folds in the clothing. Wash and rinse (using cold or warm water) until the contaminant is thoroughly removed.

Remove PPE by rolling downward (from head to toe) and avoid pulling PPE off over the head. Remove the SCBA after other PPE has been removed.

Place all PPE in labeled durable 6-mil polyethylene bags.

Decontamination of Patient/Victim:

Remove the patient/victim from the contaminated area and into the decontamination corridor.

Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag.

Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds.

Cover the patient/victim to prevent shock and loss of body heat.

Move the patient/victim to an area where emergency medical treatment can be provided.

First Aid

GENERAL INFORMATION: Initial treatment is primarily supportive of respiratory and cardiovascular function. The goal of treatment is to either prevent the conversion of methanol to toxic metabolites or to rapidly remove the toxic metabolites and correct metabolic and fluid abnormalities.

ANTIDOTE: Fomepizole and ethanol are effective antidotes against methanol toxicity. Fomepizole or ethanol should be administered as soon as possible once the patient/victim has been admitted to a medical care facility. See Long Term Implications: Medical Treatment for further instruction.

EYE:

Immediately remove the patient/victim from the source of exposure.

Immediately wash eyes with large amounts of tepid water for at least 15 minutes.

Seek medical attention immediately.

INGESTION:

Immediately remove the patient/victim from the source of exposure.

Ensure that the patient/victim has an unobstructed airway.

Do not induce vomiting (emesis).

Seek medical attention immediately.

INHALATION:

Immediately remove the patient/victim from the source of exposure.

Evaluate respiratory function and pulse.

Ensure that the patient/victim has an unobstructed airway.

If shortness of breath occurs or breathing is difficult (dyspnea), administer oxygen.

Assist ventilation as required. Always use a barrier or bag-valve-mask device.

If breathing has ceased (apnea), provide artificial respiration.

Seek medical attention immediately.

SKIN:

Immediately remove the patient/victim from the source of exposure.

See the Decontamination section for patient/victim decontamination procedures.

Seek medical attention immediately.

Long-Term Implications

MEDICAL TREATMENT: Antidotes fomepizole or ethanol should be administered intravenously as soon as possible to block the conversion of methanol to formic acid and prevent acidosis. Fomepizole is preferred as its efficacy and safety have been demonstrated, and its therapeutic dose is more easily maintained. Once the patient/victim has become acidotic, administration of fomepizole or ethanol may not provide much benefit, but they may be administered at the discretion of the physician in charge. Hemodialysis is the most effective form of treatment for an acidotic patient/victim. Folinic acid (leucovorin) should also be administered intravenously to increase the rate at which formate is metabolized into less toxic chemicals.

DELAYED EFFECTS OF EXPOSURE: The most common permanent adverse health effects following severe methanol poisoning are damage to or death of the nerve leading from the eye to the brain (optic neuropathy or atrophy), resulting in blindness; disease caused by damage to a particular region of the brain, resulting in difficulty walking and moving properly (Parkinsonism); damage to the brain caused by exposure to toxins, resulting in abnormal thought (encephalopathy); and damage to the peripheral nervous system.

EFFECTS OF CHRONIC OR REPEATED EXPOSURE: Methanol is not suspected to be a carcinogen. Chronic or repeated exposure to methanol is suspected to be a developmental toxicity risk. It is unknown whether chronic or repeated exposure to methanol is a reproductive toxicity risk. Methanol may cause birth defects of the central nervous system in humans. Chronic poisoning from repeated exposure to methanol vapor may produce inflammation of the eye (conjunctivitis), recurrent headaches, giddiness, insomnia, stomach disturbances, and visual failure. The most noted health consequences of longer-term exposure to lower levels of methanol are a broad range of effects on the eye. Inflammatory changes and irritation of the skin (dermatitis), occurs with chronic or repeated exposure to methanol.

On-Site Fatalities.

INCIDENT SITE:

Consult with the Incident Commander regarding the agent dispersed, dissemination method, level of PPE required, location, geographic complications (if any), and the approximate number of remains.

Coordinate responsibilities and prepare to enter the scene as part of the evaluation team along with the FBI HazMat Technician, local law enforcement evidence technician, and other relevant personnel.

Begin tracking remains using waterproof tags.

RECOVERY AND ON-SITE MORGUE:

Wear PPE until all remains are deemed free of contamination.

Establish a preliminary (holding) morgue.

Gather evidence, and place it in a clearly labeled impervious container. Hand any evidence over to the FBI.

Remove and tag personal effects.

Perform a thorough external evaluation and a preliminary identification check.

See the Decontamination section for decontamination procedures.

ENVIRONMENT/SPILLAGE DISPOSAL: The following methods can be used to decontaminate the environment/spillage disposal:

Do not touch or walk through the spilled agent if at all possible. However, if you must, personnel should wear the appropriate PPE during environmental decontamination. See the PPE section of this card for detailed information.

Keep combustibles (e.g., wood, paper, and oil) away from the spilled agent. Use water spray to reduce vapors or divert vapor cloud drift. Avoid allowing water runoff to contact the spilled agent.

Do not direct water at the spill or the source of the leak.

Stop the leak if it is possible to do so without risk to personnel, and turn leaking containers so that gas rather than liquid escapes.

Prevent entry into waterways, sewers, basements, or confined areas.

Isolate the area until gas has dispersed.

Ventilate the area.

EQUIPMENT: Agents can seep into the crevices of equipment making it dangerous to handle. The following methods can be used to decontaminate equipment:

Not established/determined.

Agent Properties.

Chemical Formula:

CH_3OH

Aqueous solubility:

Soluble

Boiling Point:

148.5 °F (64.7 °C)

Density:

Liquid: 0.79 g/cm^3 at 68 °F/39 °F (20 °C/4 °C)

Vapor: 1.11 (air = 1)

Flammability:

Highly flammable

Flashpoint:

54 °F (12 °C)

Ionization potential:

10.84 eV

Log $K_{benzene\text{-}water}$:

Not established/determined

Log K_{ow} (estimated):

-0.77

Melting Point:

-144°F (-97.8 °C)

Molecular Mass:

32.04

Soluble In:

Miscible with most organic solvents.

Specific Gravity:

0.79

Vapor Pressure:

96 mm Hg at 68 °F (20 °C)

127 mm Hg at 77 °F (25 °C)

ETHYLENE GLYCOL

Common Names:

1,2-Dihydroxyethane

1,2-Ethanediol

Glycol

Agent Characteristics

APPEARANCE: Clear, colorless, syrupy (viscous) liquid at room temperature. Often colored fluorescent yellow-green when used in automotive antifreeze.

DESCRIPTION: Ethylene glycol is a useful industrial compound found in many consumer products, including automotive antifreeze, hydraulic brake fluids, some stamp pad inks, ballpoint pens, solvents, paints, plastics, films, and cosmetics; it also is used as a pharmaceutical vehicle. Ethylene glycol has a sweet taste and is often accidentally or intentionally ingested. Ethylene glycol is chemically broken down in the body into toxic compounds. It and its toxic byproducts first affect the central nervous system (CNS), then the heart, and finally the kidneys. Ingestion of sufficient amounts can be fatal. Ethylene glycol is odorless; odor does not provide any warning of inhalation exposure to hazardous concentrations.

METHODS OF DISSEMINATION:

Indoor Air: Ethylene glycol can be released into indoor air as a liquid spray (aerosol), vapor, or mist.

Water: Ethylene glycol can be used to contaminate water.

Food: Ethylene glycol can be used to contaminate food.

Outdoor Air: Ethylene glycol can be released into outdoor air as a liquid spray (aerosol), vapor, or mist.

Agricultural: If ethylene glycol is released as a liquid spray (aerosol) or mist, it has the potential to contaminate agricultural products. If ethylene glycol is released as a vapor, it is unlikely to contaminate agricultural products.

ROUTES OF EXPOSURE: Systemic ethylene glycol toxicity can occur through ingestion. Breathing ethylene glycol vapors may cause eye and respiratory tract irritation but is unlikely to cause systemic toxicity. Ethylene glycol is poorly absorbed through the skin so systemic toxicity is unlikely. Eye exposure may lead to local adverse health effects but is unlikely to result in systemic toxicity.

Personal Protective Equipment.

GENERAL INFORMATION: First Responders should use a NIOSH-certified Chemical, Biological, Radiological, Nuclear (CBRN) Self Contained Breathing Apparatus (SCBA) with a Level A protective suit when entering an area with an unknown contaminant or when entering an area where the concentration of the contaminant is unknown. Level A protection should be used until monitoring results confirm the contaminant and the concentration of the contaminant.

NOTE: Safe use of protective clothing and equipment requires specific skills developed through training and experience.

LEVEL A: (RED ZONE): Select when the greatest level of skin, respiratory, and eye protection is required. This is the maximum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than the AEGL-2.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A Totally-Encapsulating Chemical Protective (TECP) suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, and a hard hat worn under the TECP suit are optional items.

LEVEL B: (RED ZONE): Select when the highest level of respiratory protection is necessary but a lesser level of skin protection is required. This is the minimum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than AEGL-2. It differs from Level A in that it incorporates a non-encapsulating, splash-protective, chemical-resistant splash suit that provides Level A protection against liquids but is not airtight.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL C: (YELLOW ZONE): Select when the contaminant and concentration of the contaminant are known and the respiratory protection criteria factors for using Air Purifying Respirators (APR) or Powered Air Purifying Respirators (PAPR) are met. This level is appropriate when decontaminating patient/victims.

A NIOSH-certified CBRN tight-fitting APR with a canister-type gas mask or CBRN PAPR for air levels greater than AEGL-2.

A NIOSH-certified CBRN PAPR with a loose-fitting face-piece, hood, or helmet and a filter or a combination organic vapor, acid gas, and particulate cartridge/filter combination or a continuous flow respirator for air levels greater than AEGL-1.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Escape mask, face shield, coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL D: (GREEN ZONE): Select when the contaminant and concentration of the contaminant are known and the concentration is below the appropriate occupational exposure limit or less than AEGL-1 for the stated duration times.

Limited to coveralls or other work clothes, boots, and gloves.

Emergency Response.

CHEMICAL DANGERS:

Ethylene glycol reacts with strong oxidants and acids.

EXPLOSION HAZARDS:

Lower explosive (flammable) limit in air (LEL), 3.2 %; upper explosive (flammable) limit in air (UEL), 15.3 %.

FIRE FIGHTING INFORMATION:

Ethylene glycol is combustible.

Extinguish fires using an agent suitable for the type of surrounding fire.

Use “alcohol” foam, dry chemical, or carbon dioxide.

Keep run-off water out of sewers and water sources.

INITIAL ISOLATION AND PROTECTIVE ACTION DISTANCES:

If a tank, rail car, or tank truck is involved in a fire, isolate it for 0.5 mi (800 m) in all directions; also, consider initial evacuation for 0.5 mi (800 m) in all directions.

This agent is not included in the DOT ERG 2004 Table of Initial Isolation and Protective Action Distances.

In the DOT ERG 2004 orange-bordered section of the guidebook, there are public safety recommendations to isolate an ethylene glycol (Guide 111) spill or leak area immediately for at least 330 ft (100 m) in all directions.

PHYSICAL DANGERS:

Vapors are heavier than air and will collect and stay in poorly-ventilated, low-lying, or confined areas (e.g., sewers, basements, and tanks).

Hazardous concentrations may develop quickly in enclosed, poorly-ventilated, or low-lying areas. Keep out of these areas. Stay upwind.

NFPA 704 Signal:

Health: 1

Flammability: 1

Reactivity: 0

Special:

Signs/Symptoms

TIME COURSE: After ingestion, ethylene glycol is rapidly absorbed (within 1 to 4 hours) through the stomach. Following absorption, 80 % or more of ethylene glycol is chemically converted by the body into toxic compounds. The course of ethylene glycol toxicity is classically divided into three broad overlapping categories of adverse health effects. Stage 1 (the neurological stage) lasts from 30 minutes to 12 hours after ingestion. Stage 2 (the cardiopulmonary stage) occurs between 12 and 24 hours after ingestion. Stage 3 (the renal stage)

occurs between 24 and 72 hours after ingestion. Adverse health effects can be delayed significantly by the co-ingestion of alcohol.

EFFECTS OF SHORT-TERM (LESS THAN 8-HOURS) EXPOSURE:

Early ethylene glycol intoxication resembles ethanol intoxication but without the characteristic odor of alcohol on the patient/victim's breath. Initial adverse health effects caused by ethylene glycol intoxication include central nervous system depression, intoxication, euphoria, stupor, and respiratory depression. Nausea and vomiting may occur as a result of gastrointestinal irritation. Severe toxicity may result in coma, loss of reflexes, seizures (uncommon), and irritation of the tissues lining the brain.

The toxic metabolic by-products of ethylene glycol metabolism cause a build-up of acid in the blood (metabolic acidosis). These toxic substances also affect the cardiopulmonary system and can cause renal failure. Metabolic acidosis commonly occurs after ethylene glycol intoxication, but absence of acidosis does not exclude ethylene glycol toxicity. Serum ethylene glycol levels do not correlate well with clinical presentation.

Untreated ethylene glycol poisoning can be fatal.

EYE EXPOSURE:

Exposure to vapors of ethylene glycol may cause irritation.

Exposure to liquid ethylene glycol may result in swelling of the eyelid and around and of the cornea, inflammation of the conjunctiva and iris, and conjunctival or corneal injury.

INGESTION EXPOSURE:

Mild to moderate, Stage 1: Reduced level of consciousness (CNS depression), euphoria, dizziness, headache, slurred speech, drowsiness, disorientation, inability to coordinate movements (ataxia), irritation and restlessness, involuntary eye movements (nystagmus), and nausea and vomiting (emesis).

Mild to moderate, Stage 2: Increased heart rate (tachycardia); abnormal or disordered heart rhythms (dysrhythmia); increased blood pressure (hypertension); and build-up of toxic breakdown products in the blood stream (metabolic acidosis), resulting in increased rate and depth of breathing (hyperventilation).

Mild to moderate, Stage 3: Effects are unusual following a mild to moderate exposure.

Severe, Stage 1: Decreased reflex responses, seizures, loss of consciousness, and coma.

Severe, Stage 2: More severe build-up of toxic breakdown products in the blood stream, resulting in increased rate and depth of breathing; heart damage, including congestive heart failure, resulting in accumulation of fluid in the lungs (pulmonary edema); lung damage, including adult respiratory distress syndrome (ARDS), resulting in a decreased oxygen supply to the body; multi-system organ failure; and death.

Severe, Stage 3: Reduced urine excretion; absence of urine excretion; and acute kidney failure, causing a build-up of toxic chemicals and chemical imbalances in the blood stream.

INHALATION EXPOSURE:

Exposure to very high levels of ethylene glycol vapors causes irritation of mucous membranes and the upper respiratory tract.

Exposure to levels of ethylene glycol concentrations higher than 80 ppm results in intolerable respiratory discomfort and cough.

SKIN EXPOSURE:

Irritation.

Decontamination

INTRODUCTION: The purpose of decontamination is to make an individual and/or their equipment safe by physically removing toxic substances quickly and effectively. Care should be taken during decontamination, because absorbed agent can be released from clothing and skin as a gas. Your Incident Commander will provide you with decontaminants specific for the agent released or the agent believed to have been released.

DECONTAMINATION CORRIDOR: The following are recommendations to protect the first responders from the release area:

Position the decontamination corridor upwind and uphill of the hot zone. The warm zone should include two decontamination corridors. One decontamination corridor is used to enter the warm zone and the other for exiting the warm zone into the cold zone. The decontamination zone for exiting should be upwind and uphill from the zone used to enter.

Decontamination area workers should wear appropriate PPE. See the PPE section of this card for detailed information.

A solution of detergent and water (which should have a pH value of at least 8 but should not exceed a pH value of 10.5) should be available for use in decontamination procedures. Soft brushes should be available to remove contamination from the PPE. Labeled, durable 6-mil polyethylene bags should be available for disposal of contaminated PPE.

INDIVIDUAL DECONTAMINATION: The following methods can be used to decontaminate an individual:

Decontamination of First Responder:

Begin washing PPE of the first responder using soap and water solution and a soft brush. Always move in a downward motion (from head to toe). Make sure to get into all areas, especially folds in the clothing. Wash and rinse (using cold or warm water) until the contaminant is thoroughly removed.

Remove PPE by rolling downward (from head to toe) and avoid pulling PPE off over the head. Remove the SCBA after other PPE has been removed.

Place all PPE in labeled durable 6-mil polyethylene bags.

Decontamination of Patient/Victim:

Remove the patient/victim from the contaminated area and into the decontamination corridor.

Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag.

Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds.

Cover the patient/victim to prevent shock and loss of body heat.

Move the patient/victim to an area where emergency medical treatment can be provided.

First Aid.

GENERAL INFORMATION: Initial treatment is primarily supportive. In the case of a large ingestion, treatment under a physician's direction within the first 30 to 60 minutes should include an attempt to aspirate stomach contents. As ethylene glycol is rapidly absorbed from the gastrointestinal (GI) tract, gastric aspiration by use of a nasogastric tube may be useful soon after large ingestions.

ANTIDOTE: Fomepizole and ethanol are effective antidotes against ethylene glycol toxicity. Fomepizole or ethanol should be administered as soon as possible once the patient/victim has been admitted to a medical care facility. See Long Term Implications: Medical Treatment for further instruction.

EYE:

Immediately remove the patient/victim from the source of exposure.

Immediately wash eyes with large amounts of tepid water for at least 15 minutes.

Seek medical attention immediately.

INGESTION:

Immediately remove the patient/victim from the source of exposure.

Ensure that the patient/victim has an unobstructed airway.

Do not induce vomiting (emesis).

Treat seizures with diazepam under a physician's direction or per local EMS protocol.

Monitor heart function, and evaluate for low blood pressure (hypotension), abnormal heart rhythms (dysrhythmias), and reduced respiratory function (respiratory depression).

Evaluate for low blood sugar (hypoglycemia), electrolyte disturbances, and low oxygen levels (hypoxia).

Seek medical attention immediately.

INHALATION:

Immediately remove the patient/victim from the source of exposure.

Evaluate respiratory function and pulse.

Ensure that the patient/victim has an unobstructed airway.

If shortness of breath occurs or breathing is difficult (dyspnea), administer oxygen.

Assist ventilation as required. Always use a barrier or bag-valve-mask device.

If breathing has ceased (apnea), provide artificial respiration.

Seek medical attention immediately.

SKIN:

Immediately remove the patient/victim from the source of exposure.

See the Decontamination section for patient/victim decontamination procedures.

Seek medical attention immediately.

See ATSDR Medical Management Guidelines for Acute Chemical Exposures, Ethylene Glycol, https://www.atsdr.cdc.gov/MHMI/mmg96.pdf, for detailed recommendations.

Long-Term Implications.

MEDICAL TREATMENT: For large ingestions of ethylene glycol, attempt to aspirate stomach (gastric) contents using a nasogastric tube, if it can be done within the first 30 to 60 minutes. In all patient/victims with known or suspected ethylene glycol poisoning, perform blood tests (CBC, blood glucose, serum electrolytes, magnesium, calcium, BUN, creatinine, lactate, ethylene glycol level, and ethanol level), arterial blood gas (ABG) levels and osmolarity, and a urinalysis. Repeat these tests as necessary to closely monitor the progression of toxic effects. Contact a medical toxicologist or a regional poison control center for assistance in evaluating the anion and osmolar gaps and to decide whether antidotal therapy, intravenous sodium bicarbonate, or hemodialysis is needed. Antidotes fomepizole or ethanol should be administered intravenously as soon as possible to block the conversion of ethylene glycol to formic acid and prevent acidosis. Fomepizole is preferred as its efficacy and safety have been demonstrated, and its therapeutic dose is more easily maintained. Once the patient/victim has become acidotic, administration of fomepizole or ethanol may not provide much benefit, but they may be administered at the discretion of the physician in charge. Folinic acid (leucovorin) should also be administered intravenously to increase the rate at which formate is metabolized into less toxic chemicals. Hemodialysis is the most effective form of treatment for an acidotic patient/victim and may be used when the blood ethylene glycol level is greater than 50 mg/dL, with severe metabolic or fluid abnormalities despite other therapeutic interventions, or in cases of kidney failure. Caution: Ethanol and fomepizole dosing must be adjusted during hemodialysis. Thiamine and pyridoxine facilitate a more rapid metabolism of ethylene glycol to non-toxic metabolites and should be given as a single dose IV (100 mg daily).

DELAYED EFFECTS OF EXPOSURE: Kidney (renal) failure can occur 24 to 72 hours after acute ethylene glycol ingestion. Some loss of kidney function may be permanent. In the absence of improvement of renal function, the patient/victim may die or require permanent hemodialysis. Injury to the nerves of the head and neck (cranial nerve palsies) may be of short-term or long-term duration. This may affect the nerves that control facial movement, eye movement and vision, hearing, and swallowing. Loss of the ability to move a body part (palsy) may occur 4 to 18 days post exposure in patient/victims with delayed treatment or inadequate or no treatment. Brain swelling (cerebral edema) causes an impaired level of consciousness and may cause generalized seizures, brain death, or permanent brain damage. Accumulation of fluid in the lungs (pulmo-

nary edema), due to heart or lung damage, may occur. Muscle inflammation (myositis) may occur.

EFFECTS OF CHRONIC OR REPEATED EXPOSURE: Ethylene glycol is not classifiable as a human carcinogen. Limited studies have not found ethylene glycol to be a carcinogen. It is not known whether chronic or repeated exposure to ethylene glycol increases the risk of reproductive toxicity or developmental toxicity. Chronic or repeated exposure to ethylene glycol may lead to irritation of the throat, mild headache, low backache, loss of consciousness, and nystagmus, all of which resolve if the source of exposure is removed.

On-Site Fatalities.

INCIDENT SITE:

Consult with the Incident Commander regarding the agent dispersed, dissemination method, level of PPE required, location, geographic complications (if any), and the approximate number of remains.

Coordinate responsibilities and prepare to enter the scene as part of the evaluation team along with the FBI HazMat Technician, local law enforcement evidence technician, and other relevant personnel.

Begin tracking remains using waterproof tags.

RECOVERY AND ON-SITE MORGUE:

Wear PPE until all remains are deemed free of contamination.

Establish a preliminary (holding) morgue.

Gather evidence, and place it in a clearly labeled impervious container.

Remove and tag personal effects.

Perform a thorough external evaluation and a preliminary identification check.

See the Decontamination section for decontamination procedures.

Decontaminate remains before they are removed from the incident site.

ENVIRONMENT/SPILLAGE DISPOSAL: The following methods can be used to decontaminate the environment/spillage disposal:

Do not touch or walk through the spilled agent if at all possible. However, if you must, personnel should wear the appropriate PPE during environmental decontamination. See the PPE section of this card for detailed information.

Keep combustibles (e.g., wood, paper, and oil) away from the spilled agent. Use water spray to reduce vapors or divert vapor cloud drift. Avoid allowing water runoff to contact the spilled agent.

Do not direct water at the spill or the source of the leak.

Stop the leak if it is possible to do so without risk to personnel, and turn leaking containers so that gas rather than liquid escapes.

Prevent entry into waterways, sewers, basements, or confined areas.

Isolate the area until gas has dispersed.

Ventilate the area.

CHAPTER 9
VOMITING AGENTS

ADAMSITE (DM)

Common Names:

10-chloro-5,10-dihydroarsacridine
10-chloro-5,10-dihydrophenarsazine
5-aza-10-arsenaanthracene chloride
Diphenylaminechlorarsine

Agent Characteristics

APPEARANCE: Light green to yellow crystals (solid) at room temperature. When dispersed by heat, fine particulate smoke; canary yellow when concentrated, colorless when diluted with air.

DESCRIPTION: Adamsite (DM) is a vomiting compound that has been used as a riot-control agent (military designation, DM). It is released as an aerosol. Adverse health effects due to exposure to adamsite (DM) are generally self-limited and do not require specific therapy. Most adverse health effects resolve within 30 minutes. Exposure to large concentrations of adamsite (DM), or exposure to adamsite (DM) within an enclosed space or under adverse weather conditions, may result in more severe adverse health effects, serious illness, or death.

METHODS OF DISSEMINATION:

Indoor Air: Adamsite (DM) can be released into indoor air as fine particles (aerosol).

Water: Adamsite (DM) is not soluble in water and cannot be used to contaminate water.

Food: Not established/determined

Outdoor Air: Adamsite (DM) can be released into outdoor air as fine particles (aerosol).

Agricultural: If adamsite (DM) is released into the air as fine particles (aerosol), it has the potential to contaminate agricultural products.

ROUTES OF EXPOSURE: Adamsite (DM) can affect the body through inhalation, ingestion, skin contact, or eye contact. Ingestion is an uncommon route of exposure.

Personal Protective Equipment

GENERAL INFORMATION: First Responders should use a NIOSH-certified Chemical, Biological, Radiological, Nuclear (CBRN) Self Contained Breathing Apparatus (SCBA) with a Level A protective suit when entering an area with an unknown contaminant or when entering an area where the concentration of the contaminant is unknown. Level A protection should be used until monitoring results confirm the contaminant and the concentration of the contaminant.

NOTE: Safe use of protective clothing and equipment requires specific skills developed through training and experience.

LEVEL A: (RED ZONE): Select when the greatest level of skin, respiratory, and eye protection is required. This is the maximum protection for workers

in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than the AEGL-2.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A Totally-Encapsulating Chemical Protective (TECP) suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, and a hard hat worn under the TECP suit are optional items.

LEVEL B: (RED ZONE): Select when the highest level of respiratory protection is necessary but a lesser level of skin protection is required. This is the minimum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than AEGL-2. It differs from Level A in that it incorporates a non-encapsulating, splash-protective, chemical-resistant splash suit that provides Level A protection against liquids but is not airtight.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL C: (YELLOW ZONE): Select when the contaminant and concentration of the contaminant are known and the respiratory protection criteria factors for using Air Purifying Respirators (APR) or Powered Air Purifying Respirators (PAPR) are met. This level is appropriate when decontaminating patient/victims.

A NIOSH-certified CBRN tight-fitting APR with a canister-type gas mask or CBRN PAPR for air levels greater than AEGL-2.

A NIOSH-certified CBRN PAPR with a loose-fitting face-piece, hood, or helmet and a filter or a combination organic vapor, acid gas, and particulate cartridge/filter combination or a continuous flow respirator for air levels greater than AEGL-1.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Escape mask, face shield, coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL D: (GREEN ZONE): Select when the contaminant and concentration of the contaminant are known and the concentration is below the appropriate occupational exposure limit or less than AEGL-1 for the stated duration times.

Limited to coveralls or other work clothes, boots, and gloves.

Emergency Response.

CHEMICAL DANGERS:

Adamsite (DM) corrodes iron, bronze, and brass.

Contact with metals may evolve flammable hydrogen gas.

EXPLOSION HAZARDS:

Containers may explode when heated.

FIRE FIGHTING INFORMATION:

Adamsite (DM) is non-combustible.

The agent itself does not burn, but it may decompose upon heating to produce corrosive and/or toxic fumes.

Fire may produce irritating, corrosive, and/or toxic gases.

The agent may be an oxidant, and it may ignite combustibles (wood, paper, oil, clothing, etc.).

For small fires, use dry chemical, carbon dioxide, or water spray.

For large fires, use dry chemical, carbon dioxide, alcohol-resistant foam, or water spray. Move containers from the fire area if it is possible to do so without risk to personnel. Dike fire control water for later disposal; do not scatter the material.

For fire involving tanks or car/trailer loads, fight the fire from maximum distance or use unmanned hose holders or monitor nozzles. Do not get water inside containers. Cool containers with flooding quantities of water until well after the fire is out. Withdraw immediately in case of rising sound from venting safety devices or discoloration of tanks. Always stay away from tanks engulfed in fire.

Run-off from fire control or dilution water may be corrosive and/or toxic, and it may cause pollution.

If the situation allows, control and properly dispose of run-off (effluent).

INITIAL ISOLATION AND PROTECTIVE ACTION DISTANCES:

If a tank, rail car, or tank truck is involved in a fire, isolate it for 0.5 mi (800 m) in all directions; also consider initial evacuation for 0.5 mi (800 m) in all directions.

Small spills (involving the release of approximately 52.83 gallons (200 liters) or less), when used as a weapon

First isolate in all directions: 200 ft (60 m).

Then protect persons downwind during the day: 0.2 mi (0.4 km).

Then protect persons downwind during the night: 0.7 mi (1.2 km).

Large spills (involving quantities greater than 52.83 gallons (200 liters)), when used as a weapon

First isolate in all directions: 600 ft (180 m).

Then protect persons downwind during the day: 1.4 mi (2.3 km).

Then protect persons downwind during the night: 3.2 mi (5.2 km).

PHYSICAL DANGERS:

Aerosol may collect and stay in confined areas (e.g., sewers, basements, and tanks).

Hazardous concentrations may develop quickly in enclosed, poorly-ventilated, or low-lying areas. Keep out of these areas. Stay upwind.

NFPA 704 Signal:

Health: 2

Flammability: 1

Reactivity: 0

Special:

Signs/Symptoms

TIME COURSE: The short-term effects of adamsite (DM) exposure begin 2 to 4 minutes after the onset of exposure. Prolonged whole-body (systemic) effects may last for 1 to 2 hours. Inhalation of DM in an outdoor environment results in adverse health effects that usually resolve in 20 minutes to 2 hours, leaving no residual effects. Inhalation of DM in an indoor environment may produce serious illness or death.

EFFECTS OF SHORT-TERM (LESS THAN 8-HOURS) EXPOSURE: Exposure to adamsite (DM) causes rapid onset irritation of the eyes, upper airway, and skin and also nausea and vomiting (emesis). Whole-body (systemic) effects are more prolonged, lasting for several hours after exposure. If concentrations are low, initial symptoms may resemble those of a severe cold.

EYE EXPOSURE:

Irritation and burning, tear production (lacrimation), spasmodic blinking (blepharospasm), swelling of the blood vessels that supply the membranes lining the eye (conjunctival injection), necrosis of the corneal epithelium.

INGESTION EXPOSURE:

Not established/determined

INHALATION EXPOSURE:

Short-term effects: Upper respiratory tract irritation in nose and sinuses, burning in throat, chest tightness and pain, uncontrollable and violent coughing and sneezing, greatly increased nasal secretions and oral secretions.

Whole-body (systemic) effects: Nausea, vomiting (emesis), abdominal cramps, diarrhea, feeling of generalized weakness (malaise), headache, mental depression, and chills.

SKIN EXPOSURE:

Dermal exposures to low concentrations of adamsite (DM) are likely to produce short-lived skin redness (erythema) and irritation. Exposure to higher

concentrations of adamsite (DM) can result in more severe, longer-lasting redness, itching, and swelling possibly followed by blister (vesicle) formation. More severe skin irritation may require symptomatic treatment.

Decontamination.

INTRODUCTION: The purpose of decontamination is to make an individual and/or their equipment safe by physically removing toxic substances quickly and effectively. Care should be taken during decontamination, because absorbed agent can be released from clothing and skin as a gas. Your Incident Commander will provide you with decontaminants specific for the agent released or the agent believed to have been released.

DECONTAMINATION CORRIDOR: The following are recommendations to protect the first responders from the release area:

Position the decontamination corridor upwind and uphill of the hot zone.

The warm zone should include two decontamination corridors. One decontamination corridor is used to enter the warm zone and the other for exiting the warm zone into the cold zone. The decontamination zone for exiting should be upwind and uphill from the zone used to enter.

Decontamination area workers should wear appropriate PPE. See the PPE section of this card for detailed information.

A solution of detergent and water (which should have a pH value of at least 8 but should not exceed a pH value of 10.5) should be available for use in decontamination procedures. Soft brushes should be available to remove contamination from the PPE.

Labeled, durable 6-mil polyethylene bags should be available for disposal of contaminated PPE.

INDIVIDUAL DECONTAMINATION: The following methods can be used to decontaminate an individual:

Decontamination of First Responder:

Begin washing PPE of the first responder using soap and water solution and a soft brush. Always move in a downward motion (from head to toe). Make sure to get into all areas, especially folds in the clothing. Wash and rinse (using cold or warm water) until the contaminant is thoroughly removed.

Remove PPE by rolling downward (from head to toe) and avoid pulling PPE off over the head. Remove the SCBA after other PPE has been removed.

Place all PPE in labeled durable 6-mil polyethylene bags.

Decontamination of Patient/Victim:

Remove the patient/victim from the contaminated area and into the decontamination corridor.

Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag.

Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to

break the patient/victim's skin during the decontamination process, and cover all open wounds.

Cover the patient/victim to prevent shock and loss of body heat.

Move the patient/victim to an area where emergency medical treatment can be provided.

First Aid.

GENERAL INFORMATION: The effects of exposure to vomiting agents under usual outdoor conditions generally are self-limited, disappearing in 20 minutes to 2 hours, and require no specific therapy other than symptomatic relief. Exposure to large concentrations of adamsite (DM), or exposure to adamsite (DM) within an enclosed space or under adverse weather conditions, may result in more severe adverse health effects, serious illness, or death and may require supportive measures for symptomatic complaints of eye, skin, and airway irritation.

ANTIDOTE: There is no antidote for adamsite (DM) toxicity.

EYE:

Immediately remove the patient/victim from the source of exposure.

Immediately wash eyes with large amounts of tepid water for at least 15 minutes.

Provide symptomatic relief.

Seek medical attention immediately.

INGESTION:

Immediately remove the patient/victim from the source of exposure.

Provide symptomatic relief.

Seek medical attention immediately.

INHALATION:

Immediately remove the patient/victim from the source of exposure.

Evaluate respiratory function and pulse.

Ensure that the patient/victim has an unobstructed airway.

If shortness of breath occurs or breathing is difficult (dyspnea), administer oxygen.

Assist ventilation as required. Always use a barrier or bag-valve-mask device.

If breathing has ceased (apnea), provide artificial respiration.

Gargling may provide symptomatic relief.

Seek medical attention immediately.

SKIN:

Immediately remove the patient/victim from the source of exposure.

May require the use of soothing compounds such as calamine, camphor, or mentholated creams.

See the Decontamination section for patient/victim decontamination procedures.

Seek medical attention immediately.

Long-Term Implications

MEDICAL TREATMENT: A small minority (fewer than 1%) of people will experience serious, prolonged adverse health effects following adamsite exposure. Those seeking medical attention will generally have complaints relating

to ocular, airway, and/or skin irritation. Patient/victims with severe or prolonged adverse health effects should be observed until effects abate.

Eyes should be carefully examined for retained foreign bodies and irrigated with water or saline. Use of topical antibiotics and eye solutions to relieve irritation should be used and referral to an ophthalmologist is suggested.

Use of oxygen and bronchodilators (if bronchospasm is present) may be necessary in patients with underlying respiratory disease.

More persistent and severe skin irritation may require the use of soothing compounds such as calamine, camphor, or mentholated creams. Large blisters, if present, should be debrided and irrigated several times daily. Use of a topical dermal antibiotic is recommended.

DELAYED EFFECTS OF EXPOSURE: Not established/determined.

EFFECTS OF CHRONIC OR REPEATED EXPOSURE: Information is unavailable about the carcinogenicity, developmental toxicity, or reproductive toxicity of chronic or repeated exposure to adamsite (DM).

On-Site Fatalities.

INCIDENT SITE:

Consult with the Incident Commander regarding the agent dispersed, dissemination method, level of PPE required, location, geographic complications (if any), and the approximate number of remains.

Coordinate responsibilities and prepare to enter the scene as part of the evaluation team along with the FBI HazMat Technician, local law enforcement evidence technician, and other relevant personnel.

Begin tracking remains using waterproof tags.

RECOVERY AND ON-SITE MORGUE:

Wear PPE until all remains are deemed free of contamination.

Establish a preliminary (holding) morgue.

Gather evidence, and place it in a clearly labeled impervious container.

Remove and tag personal effects.

Perform a thorough external evaluation and a preliminary identification check.

See the Decontamination section for decontamination procedures.

Decontaminate remains before they are removed from the incident site.

Decontamination (Environment and Equipment)

ENVIRONMENT/SPILLAGE DISPOSAL: The following methods can be used to decontaminate the environment/spillage disposal:

Do not touch or walk through the spilled agent if at all possible. However, if you must, personnel should wear the appropriate PPE during environmental decontamination. See the PPE section of this card for detailed information.

Keep combustibles (e.g., wood, paper, and oil) away from the spilled agent.

Use water spray to reduce vapors or divert vapor cloud drift. Avoid allowing water runoff to contact the spilled agent.

Do not direct water at the spill or the source of the leak.

Stop the leak if it is possible to do so without risk to personnel, and turn leaking containers so that gas rather than liquid escapes.

Prevent entry into waterways, sewers, basements, or confined areas.

Isolate the area until gas has dispersed.

Ventilate the area.

Agent Properties

Chemical Formula:

$C_{12}H_9AsClN$

Aqueous solubility:

Slightly soluble

Boiling Point:

770 °F (410 °C) (decomposes)

Density:

Solid: 1.65

Vapor: Negligible

Flammability:

Not combustible

Flashpoint:

Does not flash

Ionization potential:

Not established/determined

Log $K_{benzene\text{-}water}$:

Not established/determined

Log K_{ow} (estimated):

4.05

Melting Point:

383 °F (195 °C)

Molecular Mass:

277.59

Soluble In:

Slightly soluble in benzene, xylene, carbon tetrachloride

Specific Gravity:

Not established/determined

Vapor Pressure:

2×10^{-13} mm Hg at 68 °F (20 °C)

Volatility:

19,300 mg/m^3 at 32 °F (0 °C)

26,000 to 120,000 mg/m^3 at 68 °F (20 °C)

72,500 to 143,000 mg/m^3 at 77 °F (25 °C)

CHAPTER 10
INCAPACITATING AGENTS

FENTANYL

Agent Characteristics

APPEARANCE: Crystals or crystalline powder.

DESCRIPTION: Fentanyl is a member of the class of drugs known as fentanyls, rapid-acting opioid (synthetic opiate) drugs that alleviate pain without causing loss of consciousness (analgesic). Fentanyl depresses central nervous system (CNS) and respiratory function. Exposure to fentanyl may be fatal. Fentanyl is estimated to be 80 times as potent as morphine and hundreds of times more potent than heroin. It is a drug of abuse. Fentanyl (and other opioids) could possibly be used as an incapacitating agent to impair a person's ability to function. In October 2002, the Russian military reportedly used "a fentanyl derivative" against terrorists holding hostages in a Moscow theater; 127 of the hostages died. (It is unclear whether the gas used also included other chemical agent(s).) Fentanyl is odorless.

METHODS OF DISSEMINATION:

Indoor Air: Fentanyl can be released into indoor air as fine particles or liquid spray (aerosol).

Water: Fentanyl can be used to contaminate water.

Food: Fentanyl can be used to contaminate food.

Outdoor Air: Fentanyl can be released into outdoor air as fine particles or liquid spray (aerosol).

Agricultural: If fentanyl is released into the air as fine particles or liquid spray (aerosol), it has the potential to contaminate agricultural products.

ROUTES OF EXPOSURE: Fentanyl can be absorbed into the body via inhalation, oral exposure or ingestion, or skin contact. It is not known whether fentanyl can be absorbed systemically through the eye. Fentanyl can be administered intravenously (IV), intramuscularly (IM), or as a skin patch (transdermally).

Personal Protective Equipment

GENERAL INFORMATION: First Responders should use a NIOSH-certified Chemical, Biological, Radiological, Nuclear (CBRN) Self Contained Breathing Apparatus (SCBA) with a Level A protective suit when entering an area with an unknown contaminant or when entering an area where the concentration of the contaminant is unknown. Level A protection should be used until monitoring results confirm the contaminant and the concentration of the contaminant.

NOTE: Safe use of protective clothing and equipment requires specific skills developed through training and experience.

LEVEL A: (RED ZONE): Select when the greatest level of skin, respiratory, and eye protection is required. This is the maximum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than the AEGL-2.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A Totally-Encapsulating Chemical Protective (TECP) suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, and a hard hat worn under the TECP suit are optional items.

LEVEL B: (RED ZONE): Select when the highest level of respiratory protection is necessary but a lesser level of skin protection is required. This is the minimum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than AEGL-2. It differs from Level A in that it incorporates a non-encapsulating, splash-protective, chemical-resistant splash suit that provides Level A protection against liquids but is not airtight.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL C: (YELLOW ZONE): Select when the contaminant and concentration of the contaminant are known and the respiratory protection criteria factors for using Air Purifying Respirators (APR) or Powered Air Purifying Respirators (PAPR) are met. This level is appropriate when decontaminating patient/victims.

A NIOSH-certified CBRN tight-fitting APR with a canister-type gas mask or CBRN PAPR for air levels greater than AEGL-2.

A NIOSH-certified CBRN PAPR with a loose-fitting face-piece, hood, or helmet and a filter or a combination organic vapor, acid gas, and particulate cartridge/filter combination or a continuous flow respirator for air levels greater than AEGL-1.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Escape mask, face shield, coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL D: (GREEN ZONE): Select when the contaminant and concentration of the contaminant are known and the concentration is below the appropriate occupational exposure limit or less than AEGL-1 for the stated duration times.

Limited to coveralls or other work clothes, boots, and gloves.

Emergency Response

CHEMICAL DANGERS:

Hazardous polymerization will not occur.

EXPLOSION HAZARDS:

Not established/determined

FIRE FIGHTING INFORMATION:

Burning may produce carbon monoxide, carbon dioxide, and nitrogen oxides.

INITIAL ISOLATION AND PROTECTIVE ACTION DISTANCES:

If a tank, rail car, or tank truck is involved in a fire, isolate it for 0.5 mi (800 m) in all directions; also consider initial evacuations for 0.5 mi (800 m) in all directions.

This agent is not included in the DOT ERG 2004 Table of Initial Isolation and Protective Action Distances.

In the DOT ERG 2004 orange-bordered section of the guidebook, there are public safety recommendations to isolate a fentanyl (Guide 111) spill or leak area immediately for at least 330 ft (100 m) in all directions.

PHYSICAL DANGERS:

Not established/determined

NFPA 704 Signal:

Health: 4

Flammability: 1

Reactivity: 0

Special:

Signs/Symptoms

TIME COURSE: Peak analgesia occurs within several minutes of intravenous (IV) administration. The duration of analgesia is 30 to 60 minutes after a single dose of up to 100 µg. Dermal exposure to fentanyl results in absorption over hours to days. Oral exposure occurs in two phases. Initial exposure will occur within in a few minutes, with absorption through the intestinal tract occurring over 2 hours. Inhalation of fentanyl results in rapid absorption.

EFFECTS OF SHORT-TERM (LESS THAN 8-HOURS) EXPOSURE: Fentanyl can produce delayed reduced respiratory function (respiratory depression) and respiratory arrest. With rapid intravenous (IV) administration, rigidity of the

chest muscles («wooden chest syndrome») may be produced, which interferes with normal breathing. A rise of blood pressure within the brain (intracranial hypertension) and muscle rigidity and spasms have been reported following fentanyl use.

EYE EXPOSURE:

Irritation may occur.

INGESTION EXPOSURE:

Contracted or pinpoint pupils (miosis) (may later become dilated), reduced level of consciousness (CNS depression), reduced respiratory function (respiratory depression), reduced blood oxygen content (hypoxia), accumulation of acid in the blood (acidosis), low blood pressure (hypotension), slow heart rate (bradycardia), shock, slowing of muscular movement of the stomach (gastric hypomotility) with intestinal obstruction due to lack of normal muscle function (ileus), accumulation of fluid in the lungs (pulmonary edema), lethargy, coma, and death.

INHALATION EXPOSURE:

See Ingestion Exposure.

SKIN EXPOSURE:

See Ingestion Exposure.

Absorption through the skin may contribute to whole-body (systemic) toxicity.

Absorption increases with skin temperature (based on medical use of transdermal patch).

Decontamination.

INTRODUCTION: The purpose of decontamination is to make an individual and/or their equipment safe by physically removing toxic substances quickly and effectively. Care should be taken during decontamination, because absorbed agent can be released from clothing and skin as a gas. Your Incident Commander will provide you with decontaminants specific for the agent released or the agent believed to have been released.

DECONTAMINATION CORRIDOR: The following are recommendations to protect the first responders from the release area:

Position the decontamination corridor upwind and uphill of the hot zone. The warm zone should include two decontamination corridors. One decontamination corridor is used to enter the warm zone and the other for exiting the warm zone into the cold zone. The decontamination zone for exiting should be upwind and uphill from the zone used to enter.

Decontamination area workers should wear appropriate PPE. See the PPE section of this card for detailed information.

A solution of detergent and water (which should have a pH value of at least 8 but should not exceed a pH value of 10.5) should be available for use in decontamination procedures. Soft brushes should be available to remove contamination from the PPE. Labeled, durable 6-mil polyethylene bags should be available for disposal of contaminated PPE.

INDIVIDUAL DECONTAMINATION: The following methods can be used to decontaminate an individual:

Decontamination of First Responder:

Begin washing PPE of the first responder using soap and water solution and a soft brush. Always move in a downward motion (from head to toe). Make sure to get into all areas, especially folds in the clothing. Wash and rinse (using cold or warm water) until the contaminant is thoroughly removed.

Remove PPE by rolling downward (from head to toe) and avoid pulling PPE off over the head. Remove the SCBA after other PPE has been removed.

Place all PPE in labeled durable 6-mil polyethylene bags.

Decontamination of Patient/Victim:

Remove the patient/victim from the contaminated area and into the decontamination corridor.

Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag.

Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds.

Cover the patient/victim to prevent shock and loss of body heat.

Move the patient/victim to an area where emergency medical treatment can be provided.

First Aid

GENERAL INFORMATION: Treatment consists of administration of the antidote and aggressive support of respiratory function.

ANTIDOTE: Naloxone (Narcan) in doses of 0.4 to 2.0 mg has been recommended for treatment of opioid overdose. Naloxone is commonly given intravenously. The onset of effect following IV naloxone administration is 1 to 3 minutes; maximal effect is observed within 5 to 10 minutes. Doses may be repeated as needed to maintain effect. Administration of naloxone may also reverse the «wooden chest syndrome».

EYE:

Immediately remove the patient/victim from the source of exposure.

Immediately wash eyes with large amounts of tepid water for at least 15 minutes.

Seek medical attention immediately.

INGESTION:

Immediately remove the patient/victim from the source of exposure.

Ensure that the patient/victim has an unobstructed airway.

Do not induce vomiting (emesis).

Administer naloxone under physician's direction or by following applicable EMS protocol. See Antidote section.

Administer charcoal as a slurry (240 mL water/30 g charcoal). Usual dose: 25 to 100 g in adults/adolescents, 25 to 50 g in children (1 to 12 years), and 1 g/kg in infants less than 1 year old.

Seek medical attention immediately.

INHALATION:

Immediately remove the patient/victim from the source of exposure.

Evaluate respiratory function and pulse.

Ensure that the patient/victim has an unobstructed airway.

If shortness of breath occurs or breathing is difficult (dyspnea), administer oxygen.

Assist ventilation as required. Always use a barrier or bag-valve-mask device.

If breathing has ceased (apnea), provide artificial respiration.

Monitor the patient/victim for signs of whole-body (systemic) effects and administer symptomatic treatment as necessary.

If signs of whole-body (systemic) poisoning appear, see the Ingestion section for treatment recommendations.

Seek medical attention immediately.

SKIN:

Immediately remove the patient/victim from the source of exposure.

See the Decontamination section for patient/victim decontamination procedures.

Monitor the patient/victim for signs of whole-body (systemic) effects.

If signs of whole-body (systemic) poisoning appear, see the Ingestion section for treatment recommendations.

Seek medical attention immediately.

Long-Term Implications

MEDICAL TREATMENT: Patient/victims exhibiting significantly reduced respiratory function (respiratory depression), recurrent sedation, or any other complicating factors of opioid toxicity should be admitted for a minimum of 12 to 24 hours of observation. Heart function should be monitored, and the patient/victim should be evaluated for low blood pressure (hypotension), abnormal heart rhythms (dysrhythmias), and reduced respiratory function (respiratory depression). Accumulation of fluid in the lungs (pulmonary edema) is a common aftereffect (sequela), and patient/victims should be monitored for its development and treated accordingly.

DELAYED EFFECTS OF EXPOSURE: Not established/determined.

EFFECTS OF CHRONIC OR REPEATED EXPOSURE: It is unknown whether chronic or repeated exposure to fentanyl increases the risk of carcinogenicity, reproductive toxicity, or developmental toxicity.

On-Site Fatalities.

INCIDENT SITE:

Consult with the Incident Commander regarding the agent dispersed, dissemination method, level of PPE required, location, geographic complications (if any), and the approximate number of remains.

Coordinate responsibilities and prepare to enter the scene as part of the evaluation team along with the FBI HazMat Technician, local law enforcement evidence technician, and other relevant personnel.

Begin tracking remains using waterproof tags.

RECOVERY AND ON-SITE MORGUE:

Wear PPE until all remains are deemed free of contamination.

Establish a preliminary (holding) morgue.

Gather evidence, and place it in a clearly labeled impervious container.

Remove and tag personal effects.

Perform a thorough external evaluation and a preliminary identification check.

See the Decontamination section for decontamination procedures.

Decontaminate remains before they are removed from the incident site.

ENVIRONMENT/SPILLAGE DISPOSAL: The following methods can be used to decontaminate the environment/spillage disposal:

Do not touch or walk through the spilled agent if at all possible. However, if you must, personnel should wear the appropriate PPE during environmental decontamination. See the PPE section of this card for detailed information.

Keep combustibles (e.g., wood, paper, and oil) away from the spilled agent. Use water spray to reduce vapors or divert vapor cloud drift. Avoid allowing water runoff to contact the spilled agent.

Do not direct water at the spill or the source of the leak.

Stop the leak if it is possible to do so without risk to personnel, and turn leaking containers so that gas rather than liquid escapes.

Prevent entry into waterways, sewers, basements, or confined areas.

Isolate the area until gas has dispersed.

Ventilate the area.

EQUIPMENT: Agents can seep into the crevices of equipment making it dangerous to handle. The following methods can be used to decontaminate equipment:

Not established/determined

Agent Properties

Chemical Formula:

$C_{22}H_{28}N_2O$

Aqueous solubility:

Slightly soluble

Boiling Point:

Not established/determined

Density:

Not established/determined

Flammability:

Not established/determined

Flashpoint:

Not established/determined

Ionization potential:

Not established/determined

Log $K_{benzene\text{-}water}$:

Not established/determined

Log K_{ow} (estimated): 4.05

Melting Point:

181.4° to 183.2 °F (83° to 84 °C)

Molecular Mass: 336.47

CHAPTER 11
BIOTOXINS

TETRODOTOXIN

Common Names:

Fugu poison
Maculotoxin
Spheroidine
Tarichatoxin
Tetrodontoxin
TTX

Agent Characteristics

APPEARANCE: Colorless crystalline solid that darkens when heated above 428 °F (220 °C).

DESCRIPTION: Tetrodotoxin is an extremely potent poison (toxin) found mainly in the liver and sex organs (gonads) of some fish, such as puffer fish, globefish, and toadfish (order Tetraodontiformes) and in some amphibian, octopus, and shellfish species. Human poisonings occur when the flesh and/or organs of the fish are improperly prepared and eaten. Tetrodotoxin interferes with the transmission of signals from nerves to muscles and causes an increasing paralysis of the muscles of the body. Tetrodotoxin poisoning can be fatal.

METHODS OF DISSEMINATION:

Indoor Air: Because the natural source of tetrodotoxin is from living organisms, and manufacturing tetrodotoxin artificially in appreciable quantities is extremely difficult, dissemination through indoor air is unlikely.

Water: Because the natural source of tetrodotoxin is from living organisms, and manufacturing tetrodotoxin artificially in appreciable quantities is extremely difficult, dissemination through water is unlikely.

Food: Exposure to tetrodotoxin usually occurs through eating improperly prepared fish or possibly through contamination of other food products.

Outdoor Air: Because the natural source of tetrodotoxin is from living organisms, and manufacturing tetrodotoxin artificially in appreciable quantities is extremely difficult, dissemination through outdoor air is unlikely.

Agricultural: Because tetrodotoxin is unlikely to be disseminated through indoor or outdoor air, it is also unlikely to be disseminated through agricultural products.

ROUTES OF EXPOSURE: Exposure occurs due to ingestion of fish or other food containing tetrodotoxin.

Personal Protective Equipment

GENERAL INFORMATION: The following are general recommendations for hazardous material exposure. However, it is unlikely that tetrodotoxin will require this amount of protection.

First Responders should use a NIOSH-certified Chemical, Biological, Radiological, Nuclear (CBRN) Self Contained Breathing Apparatus (SCBA) with a Level A protective suit when entering an area with an unknown contaminant or when entering an area where the concentration of the contaminant is unknown. Level A protection should be used until monitoring results confirm the contaminant and the concentration of the contaminant.

NOTE: Safe use of protective clothing and equipment requires specific skills developed through training and experience.

LEVEL A: (RED ZONE): Select when the greatest level of skin, respiratory, and eye protection is required. This is the maximum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than the AEGL-2.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A Totally-Encapsulating Chemical Protective (TECP) suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, and a hard hat worn under the TECP suit are optional items.

LEVEL B: (RED ZONE): Select when the highest level of respiratory protection is necessary but a lesser level of skin protection is required. This is the minimum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than AEGL-2. It differs from Level A in that it incorporates a non-encapsulating, splash-protective, chemical-resistant splash suit that provides Level A protection against liquids but is not airtight.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL C: (YELLOW ZONE): Select when the contaminant and concentration of the contaminant are known and the respiratory protection criteria factors for using Air Purifying Respirators (APR) or Powered Air Purifying Respirators (PAPR) are met. This level is appropriate when decontaminating patient/victims.

A NIOSH-certified CBRN tight-fitting APR with a canister-type gas mask or CBRN PAPR for air levels greater than AEGL-2.

A NIOSH-certified CBRN PAPR with a loose-fitting face-piece, hood, or helmet and a filter or a combination organic vapor, acid gas, and particulate cartridge/filter combination or a continuous flow respirator for air levels greater than AEGL-1.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Escape mask, face shield, coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL D: (GREEN ZONE): Select when the contaminant and concentration of the contaminant are known and the concentration is below the appropriate occupational exposure limit or less than AEGL-1 for the stated duration times.

Limited to coveralls or other work clothes, boots, and gloves.

Emergency Response

CHEMICAL DANGERS:

Not established/determined

EXPLOSION HAZARDS:

Not established/determined

FIRE FIGHTING INFORMATION:

Not established/determined

INITIAL ISOLATION AND PROTECTIVE ACTION DISTANCES:

Not applicable

PHYSICAL DANGERS:

Not established/determined

NFPA 704 Signal:

Health: 4

Flammability: 1

Reactivity: 0

Special:

Signs/Symptoms

TIME COURSE: Tetrodotoxin poisoning may either have rapid onset (10 to 45 minutes) or delayed onset (generally within 3 to 6 hours but rarely longer). Death may occur as early as 20 minutes, or as late as 24 hours, after exposure;

but it usually occurs within the first 4 to 8 hours. Patient/victims who live through the acute intoxication in the first 24 hours usually recover without residual deficits. Symptoms may last for several days and recovery takes days to occur.

EFFECTS OF SHORT-TERM (LESS THAN 8-HOURS) EXPOSURE: Tetrodotoxin interferes with the transmission of signals from nerves to muscles by blocking sodium channels. This results in rapid weakening and paralysis of muscles, including those of the respiratory tract, which can lead to respiratory arrest and death.

EYE EXPOSURE:

Not established/determined

INGESTION EXPOSURE:

First stage: Numbness and sensation of prickling and tingling (paresthesia) of the lips and tongue, followed by facial and extremity paresthesias and numbness, headache, sensations of lightness or floating, profuse sweating (diaphoresis), dizziness, salivation (ptyalism), nausea, vomiting (emesis), diarrhea, abdominal (epigastric) pain, difficulty moving (motor dysfunction), weakness (malaise), and speech difficulties.

Second stage: Increasing paralysis, first in the extremities, then in the rest of the body, and finally in the respiratory muscles; difficulty breathing or shortness of breath (dyspnea); abnormal heart rhythms (cardiac dysrhythmias or arrhythmia); abnormally low blood pressure (hypotension); fixed and dilated pupils (mydriasis); coma; seizures; respiratory arrest; and death.

INHALATION EXPOSURE:

Not established/determined

SKIN EXPOSURE:

Not established/determined

Decontamination

INTRODUCTION: The following are general recommendations for hazardous material exposure. However, it is unlikely that tetrodotoxin will require extensive decontamination, as its toxicity is primarily through individual ingestion. Off gassing is not believed to be a hazard. Safe disposal of the contaminated food and cleaning of all food preparation surfaces and utensils with soap and water should be sufficient.

The purpose of decontamination is to make an individual and/or their equipment safe by physically removing toxic substances quickly and effectively. Care should be taken during decontamination, because absorbed agent can be released from clothing and skin as a gas. Your Incident Commander will provide you with decontaminants specific for the agent released or the agent believed to have been released.

DECONTAMINATION CORRIDOR: The following are recommendations to protect the first responders from the release area:

Position the decontamination corridor upwind and uphill of the hot zone.

The warm zone should include two decontamination corridors. One decontamination corridor is used to enter the warm zone and the other for exiting the

warm zone into the cold zone. The decontamination zone for exiting should be upwind and uphill from the zone used to enter.

Decontamination area workers should wear appropriate PPE. See the PPE section of this card for detailed information.

A solution of detergent and water (which should have a pH value of at least 8 but should not exceed a pH value of 10.5) should be available for use in decontamination procedures. Soft brushes should be available to remove contamination from the PPE.

Labeled, durable 6-mil polyethylene bags should be available for disposal of contaminated PPE.

INDIVIDUAL DECONTAMINATION: The following methods can be used to decontaminate an individual:

Decontamination of First Responder:

Begin washing PPE of the first responder using soap and water solution and a soft brush. Always move in a downward motion (from head to toe). Make sure to get into all areas, especially folds in the clothing. Wash and rinse (using cold or warm water) until the contaminant is thoroughly removed.

Remove PPE by rolling downward (from head to toe) and avoid pulling PPE off over the head. Remove the SCBA after other PPE has been removed.

Place all PPE in labeled durable 6-mil polyethylene bags.

Decontamination of Patient/Victim:

Remove the patient/victim from the contaminated area and into the decontamination corridor.

Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag.

Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds.

Cover the patient/victim to prevent shock and loss of body heat.

Move the patient/victim to an area where emergency medical treatment can be provided.

First Aid

GENERAL INFORMATION: Initial treatment is primarily supportive.

ANTIDOTE: There is no antidote for tetrodotoxin toxicity.

EYE:

Not established/determined

INGESTION:

Remove the patient/victim from the source of exposure.

Prevent others from eating until the source of tetrodotoxin exposure can be ascertained, in order to avoid more casualties.

Do not induce vomiting (emesis).

Administer supplemental oxygen and assist ventilation as needed.

Seek medical attention immediately.

INHALATION:

Not established/determined

SKIN:

Not established/determined

Long-Term Implications

MEDICAL TREATMENT: If the patient/victim can be rapidly transported to an emergency department following decontamination, stomach pumping (gastric lavage) may be considered after the airway has been secured. Gastric lavage is recommended only after ingestion of a life-threatening amount of tetrodotoxin and only if it can be done shortly after ingestion (generally within 1 hour). The risk of worsening injury to the lining of the gastrointestinal (GI) tract must be considered. Heart function should be monitored, and the patient/victim should be evaluated for low blood pressure (hypotension), abnormal heart rhythms (dysrhythmias), and reduced respiratory function (respiratory depression). The patient/victim should be evaluated for low blood sugar (hypoglycemia), electrolyte disturbances, and decreased oxygen supply to the tissues (hypoxia).

DELAYED EFFECTS OF EXPOSURE: Not established/determined.

EFFECTS OF CHRONIC OR REPEATED EXPOSURE: Information is unavailable about the carcinogenicity, developmental toxicity, or reproductive toxicity from chronic or repeated exposure to tetrodotoxin.

On-Site Fatalities

INCIDENT SITE:

Consult with the Incident Commander regarding the agent dispersed, dissemination method, level of PPE required, location, geographic complications (if any), and the approximate number of remains.

Coordinate responsibilities and prepare to enter the scene as part of the evaluation team along with the FBI HazMat Technician, local law enforcement evidence technician, and other relevant personnel.

Begin tracking remains using waterproof tags.

RECOVERY AND ON-SITE MORGUE:

Wear PPE until all remains are deemed free of contamination.

Establish a preliminary (holding) morgue.

Gather evidence, and place it in a clearly labeled impervious container. Hand any evidence over to the FBI.

Remove and tag personal effects.

Perform a thorough external evaluation and a preliminary identification check.

See the Decontamination section for decontamination procedures.

Decontaminate remains before they are removed from the incident site.

ENVIRONMENT/SPILLAGE DISPOSAL: The following methods can be used to decontaminate the environment/spillage disposal:

Do not touch or walk through the spilled agent if at all possible. However, if you must, personnel should wear the appropriate PPE during environmental decontamination. See the PPE section of this card for detailed information.

Keep combustibles (e.g., wood, paper, and oil) away from the spilled agent.

Use water spray to reduce vapors or divert vapor cloud drift. Avoid allowing water runoff to contact the spilled agent.

Do not direct water at the spill or the source of the leak.

Stop the leak if it is possible to do so without risk to personnel, and turn leaking containers so that gas rather than liquid escapes.

Prevent entry into waterways, sewers, basements, or confined areas.

Isolate the area until gas has dispersed.

Ventilate the area.

EQUIPMENT: Agents can seep into the crevices of equipment making it dangerous to handle. The following methods can be used to decontaminate equipment:

Not established/determined

Agent Properties

Chemical Formula:

$C_{11}H_{17}N_3O_8$

Aqueous solubility:

Slightly soluble

Molecular Mass:

319.27

Soluble In:

Soluble in dilute acetic acid; slightly soluble in dry alcohol, ether; practically insoluble in other organic solvents.

COLCHICINE

Common Names:

Colchineos

Colcin

Agent Characteristics

APPEARANCE: Pale yellow to greenish-yellow crystals, scales, or powder; darkens when exposed to light.

DESCRIPTION: Colchicine is a highly toxic plant hormone that is used medically in the treatment of gout and in scientific research. Colchicine stops the process of cell division (it is an antimitotic agent). Exposure to colchicine can be fatal in very small doses (e.g., 7 to 65 mg). Colchicine is derived from the meadow saffron or autumn crocus plant (Colchicum autumnale), which is locally abundant in meadows throughout most of Europe and has become naturalized in parts of North America. It can also be found in the tubers of the Glory Lily (Gloriosa superba) found primarily in Florida. It is odorless or nearly so and has a very bitter taste. It is produced in tablets, granules, and ampules of sterile solution. It has uses in the production of legitimate and illicit plant production.

METHODS OF DISSEMINATION:

Indoor Air: Colchicine can be released into indoor air as fine particles or liquid spray (aerosol).

Water: Colchicine can be used to contaminate water.

Food: Colchicine can be used to contaminate food.

Outdoor Air: Colchicine can be released into outdoor air as fine particles or liquid spray (aerosol).

Agricultural: If colchicine is released into the air as fine particles or liquid spray (aerosol), it has the potential to contaminate agricultural products.

ROUTES OF EXPOSURE: Colchicine can be absorbed into the body by ingestion, inhalation, or eye contact. Colchicine can also be injected through the skin or administered intravenously. It is unknown whether colchicine can be absorbed through intact skin.

Personal Protective Equipment

GENERAL INFORMATION: First Responders should use a NIOSH-certified Chemical, Biological, Radiological, Nuclear (CBRN) Self Contained Breathing Apparatus (SCBA) with a Level A protective suit when entering an area with an unknown contaminant or when entering an area where the concentration of the contaminant is unknown. Level A protection should be used until monitoring results confirm the contaminant and the concentration of the contaminant.

NOTE: Safe use of protective clothing and equipment requires specific skills developed through training and experience.

LEVEL A: (RED ZONE): Select when the greatest level of skin, respiratory, and eye protection is required. This is the maximum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than the AEGL-2.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A Totally-Encapsulating Chemical Protective (TECP) suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, and a hard hat worn under the TECP suit are optional items.

LEVEL B: (RED ZONE): Select when the highest level of respiratory protection is necessary but a lesser level of skin protection is required. This is the minimum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than AEGL-2. It differs from Level A in that it incorporates a non-encapsulating, splash-protective, chemical-resistant splash suit that provides Level A protection against liquids but is not airtight.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL C: (YELLOW ZONE): Select when the contaminant and concentration of the contaminant are known and the respiratory protection criteria factors for using Air Purifying Respirators (APR) or Powered Air Purifying Respirators (PAPR) are met. This level is appropriate when decontaminating patient/victims.

A NIOSH-certified CBRN tight-fitting APR with a canister-type gas mask or CBRN PAPR for air levels greater than AEGL-2.

A NIOSH-certified CBRN PAPR with a loose-fitting face-piece, hood, or helmet and a filter or a combination organic vapor, acid gas, and particulate cartridge/filter combination or a continuous flow respirator for air levels greater than AEGL-1.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Escape mask, face shield, coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL D: (GREEN ZONE): Select when the contaminant and concentration of the contaminant are known and the concentration is below the appropriate occupational exposure limit or less than AEGL-1 for the stated duration times.

Limited to coveralls or other work clothes, boots, and gloves.

Emergency Response

CHEMICAL DANGERS:

When heated to decomposition, colchicine emits toxic fumes of carbon monoxide, carbon dioxide, and nitrogen oxides.

Colchicine is incompatible with strong oxidants and mineral acids.

EXPLOSION HAZARDS:

Upper and lower explosive (flammable) limits in air are not available for colchicine.

FIRE FIGHTING INFORMATION:

Colchicine is non-combustible.

The agent itself does not burn, but it may decompose upon heating to produce corrosive and/or toxic fumes.

Fire may produce irritating, corrosive, and/or toxic gases.

For small fires, use dry chemical, carbon dioxide, or water spray.

For large fires, use water spray, fog, or regular foam. Move containers from the fire area if it is possible to do so without risk to personnel. Dike fire control

water for later disposal; do not scatter the material. Use water spray or fog; do not use straight streams.

For fire involving tanks or car/trailer loads, fight the fire from maximum distance or use unmanned hose holders or monitor nozzles. Do not get water inside containers. Cool containers with flooding quantities of water until well after the fire is out. Withdraw immediately in case of rising sound from venting safety devices or discoloration of tanks. Always stay away from tanks engulfed in fire.

For massive fire, use unmanned hose holders or monitor nozzles; if this is impossible, withdraw from the area and let the fire burn.

Run-off from fire control or dilution water may be corrosive and/or toxic, and it may cause pollution.

If the situation allows, control and properly dispose of run-off (effluent).

INITIAL ISOLATION AND PROTECTIVE ACTION DISTANCES:

If a tank, rail car, or tank truck is involved in a fire, isolate it for 0.5 mi (800 m) in all directions; also consider initial evacuation for 0.5 mi (800 m) in all directions.

This agent is not included in the DOT ERG 2004 Table of Initial Isolation and Protective Action Distances.

In the DOT ERG 2004 orange-bordered section of the guidebook, there are public safety recommendations to isolate a colchicine (Guide 151) spill or leak area immediately for at least 150 ft (50 m) for liquids and 75 ft (25 m) for solids in all directions.

PHYSICAL DANGERS:

Colchicine withstands drying, storage, or boiling.

NFPA 704 Signal:

Health: 3

Flammability: 1

Reactivity: 0

Special:

Signs/Symptoms

TIME COURSE: Colchicine toxicity may be fatal. Patient/victims may be asymptomatic up to 24 hours post exposure. Onset of gastrointestinal signs and symptoms may occur as early as 2 hours and as late as 24 hours post exposure. Onset of multiple system organ failure may occur within 24 to 72 hours. Recovery may begin within 6 to 8 days post exposure.

EFFECTS OF SHORT-TERM (LESS THAN 8-HOURS) EXPOSURE: The first phase of colchicine toxicity consists of gastrointestinal symptoms: burning sensation in the throat, nausea, vomiting, diarrhea, and abdominal pain. Exces-

sive fluid loss from vomiting and diarrhea may result in hypovolemic shock. Patient/victims suffering from mild exposure may have no further adverse effects.

EYE EXPOSURE:

Eye contact with liquid colchicine may produce clouding of the cornea that clears in a few weeks.

See Ingestion Exposure.

INGESTION EXPOSURE:

Gastrointestinal phase: Nausea; loss of appetite (anorexia); abdominal pain; vomiting (emesis); profuse, watery, and bloody diarrhea; joint pain; generalized weakness; fever; rashes; and hypovolemic shock.

Multiple system organ failure phase: Hypovolemic shock due to extreme vascular damage and fluid loss through the GI tract may result in death; kidney damage resulting in low urine output and bloody urine; swollen, tender liver with elevated liver enzymes; low white blood cell counts (persisting for several days); other blood (hematologic) manifestations including bone marrow depression, low platelets, and anemia; muscular weakness; ascending paralysis that may result in respiratory failure; and loss of deep tendon reflexes. The patient/victim usually remains conscious but exhibits mental confusion or delirium and seizures. Pediatric patient/victims may experience hallucinations.

INHALATION EXPOSURE:

See Signs/Symptoms Ingestion Exposure.

SKIN EXPOSURE:

Not established/determined.

Decontamination

INTRODUCTION: The purpose of decontamination is to make an individual and/or their equipment safe by physically removing toxic substances quickly and effectively. Care should be taken during decontamination, because absorbed agent can be released from clothing and skin as a gas. Your Incident Commander will provide you with decontaminants specific for the agent released or the agent believed to have been released.

DECONTAMINATION CORRIDOR: The following are recommendations to protect the first responders from the release area:

Position the decontamination corridor upwind and uphill of the hot zone.

The warm zone should include two decontamination corridors. One decontamination corridor is used to enter the warm zone and the other for exiting the warm zone into the cold zone. The decontamination zone for exiting should be upwind and uphill from the zone used to enter.

Decontamination area workers should wear appropriate PPE. See the PPE section of this card for detailed information.

A solution of detergent and water (which should have a pH value of at least 8 but should not exceed a pH value of 10.5) should be available for use in decontamination procedures. Soft brushes should be available to remove contamination from the PPE.

Labeled, durable 6-mil polyethylene bags should be available for disposal of contaminated PPE.

INDIVIDUAL DECONTAMINATION: The following methods can be used to decontaminate an individual:

Decontamination of First Responder:

Begin washing PPE of the first responder using soap and water solution and a soft brush. Always move in a downward motion (from head to toe). Make sure to get into all areas, especially folds in the clothing. Wash and rinse (using cold or warm water) until the contaminant is thoroughly removed.

Remove PPE by rolling downward (from head to toe) and avoid pulling PPE off over the head. Remove the SCBA after other PPE has been removed.

Place all PPE in labeled durable 6-mil polyethylene bags.

Decontamination of Patient/Victim:

Remove the patient/victim from the contaminated area and into the decontamination corridor.

Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag.

Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds.

Cover the patient/victim to prevent shock and loss of body heat.

Move the patient/victim to an area where emergency medical treatment can be provided.

First Aid

GENERAL INFORMATION: Initial treatment is primarily supportive. It includes relief of abdominal pain, measures to combat shock, and establishment of adequate respiratory exchange by maintenance of an adequate airway, control of respiration, and oxygen administration.

ANTIDOTE: There is no antidote for colchicine toxicity.

EYE:

Immediately remove the patient/victim from the source of exposure.

Immediately wash eyes with large amounts of tepid water for at least 15 minutes.

Seek medical attention immediately.

INGESTION:

Immediately remove the patient/victim from the source of exposure.

Ensure that the patient/victim has an unobstructed airway.

Do not induce vomiting (emesis).

Administer charcoal as a slurry (240 mL water/30 g charcoal). Usual dose: 25 to 100 g in adults/adolescents, 25 to 50 g in children (1 to 12 years), and 1 g/kg in infants less than 1 year old.

If the patient/victim can be rapidly transported to an emergency department for decontamination, stomach pumping (gastric lavage) may be considered after

the airway has been secured. Gastric lavage is recommended only after ingestion of a life-threatening amount of the agent and only if it can be done shortly after ingestion (generally within 1 hour). The risk of worsening injury to the lining of the gastrointestinal (GI) tract must be considered.

If evidence of shock or low blood pressure (hypotension) is observed, begin intravenous (IV) fluid administration.

Also see First Aid for Inhalation Exposure.

Seek medical attention immediately.

INHALATION:

Immediately remove the patient/victim from the source of exposure.

Evaluate respiratory function and pulse.

Ensure that the patient/victim has an unobstructed airway.

If shortness of breath occurs or breathing is difficult (dyspnea), administer oxygen.

Assist ventilation as required. Always use a barrier or bag-valve-mask device.

If breathing has ceased (apnea), provide artificial respiration.

If evidence of shock or low blood pressure (hypotension) is observed, begin intravenous (IV) fluid administration.

Also see First Aid for Ingestion Exposure.

Seek medical attention immediately.

SKIN:

Immediately remove the patient/victim from the source of exposure.

See the Decontamination section for patient/victim decontamination procedures.

Seek medical attention immediately.

Long-Term Implications

MEDICAL TREATMENT: Patient/victims exposed to colchicine by ingestion should be observed for gastrointestinal symptoms for 12 hours following acute exposure. Heart function should be monitored, and the patient/victim should be evaluated for low blood pressure (hypotension), abnormal heart rhythms (dysrhythmias), and reduced respiratory function (respiratory depression). Monitor other organ systems as appropriate.

DELAYED EFFECTS OF EXPOSURE: Gastrointestinal signs and symptoms are followed by multiple system organ failure. This is marked by bone marrow suppression, whole body vascular system clotting (disseminated intravascular coagulopathy), adult respiratory distress, cardiac dysfunction (dysrhythmia, failure, and arrest), intestinal obstruction (ileus), neuromuscular abnormalities (ascending paralysis, rhabdomyolysis, neuritis, and peripheral neuropathies), kidney failure, sepsis, fever, metabolic disturbances, and mental status changes are all key features of this stage. Death during this stage can result from hypovolemic, cardiac, or septic shock. These effects are due to a combination of colchicine's direct action on organs as well as massive infection from sepsis.

Patient/victims surviving multiple system organ failure will begin to experience recovery after 6 to 8 days. Recovery is marked by an increase in white

blood cell count (rebound leukocytosis), reversible hair loss (alopecia), and return of normal organ system functions.

EFFECTS OF CHRONIC OR REPEATED EXPOSURE: Information is inconclusive about the carcinogenicity, developmental toxicity, or reproductive toxicity from chronic or repeated exposure to colchicine. Effects of chronic exposure to colchicine are mainly known from its use as a drug in human medicine. In these circumstances gastrointestinal symptoms are likely to cause discontinuation of the use of the drug, long before any other symptoms develop. Bone marrow depression with a condition marked by fever and other symptoms (agranulocytosis), persistent decrease in the number of blood platelets (thrombocytopenia), reduced white blood cell count (leukopenia), and anemia characterized by defective function of the blood-forming organs (aplastic anemia) may occur with prolonged colchicine administration. Loss of body and scalp hair (alopecia), rashes, blister-like inflammation of the skin (vesicular dermatitis), degeneration of certain nerves (peripheral neuritis or neuropathy), a break down of muscle tissue at the cellular level (myopathy), the absences of urine excretion (anuria), kidney (renal) damage, blood in the urine (hematuria), and one case of patches of purplish discoloration of the skin and mucous membranes (purpura) have also been reported with prolonged administration of colchicine.

On-Site Fatalities.

INCIDENT SITE:

Consult with the Incident Commander regarding the agent dispersed, dissemination method, level of PPE required, location, geographic complications (if any), and the approximate number of remains.

Coordinate responsibilities and prepare to enter the scene as part of the evaluation team along with the FBI HazMat Technician, local law enforcement evidence technician, and other relevant personnel.

Begin tracking remains using waterproof tags.

RECOVERY AND ON-SITE MORGUE:

Wear PPE until all remains are deemed free of contamination.

Establish a preliminary (holding) morgue.

ENVIRONMENT/SPILLAGE DISPOSAL: The following methods can be used to decontaminate the environment/spillage disposal:

Do not touch or walk through the spilled agent if at all possible. However, if you must, personnel should wear the appropriate PPE during environmental decontamination. See the PPE section of this card for detailed information.

Keep combustibles (e.g., wood, paper, and oil) away from the spilled agent.

Use water spray to reduce vapors or divert vapor cloud drift. Avoid allowing water runoff to contact the spilled agent.

Do not direct water at the spill or the source of the leak.

Stop the leak if it is possible to do so without risk to personnel, and turn leaking containers so that gas rather than liquid escapes.

Prevent entry into waterways, sewers, basements, or confined areas.

Isolate the area until gas has dispersed.

Ventilate the area.

EQUIPMENT: Agents can seep into the crevices of equipment making it dangerous to handle. The following methods can be used to decontaminate equipment:

Not established/determined

Agent Properties

Chemical Formula:

$C_{22}H_{25}NO_6$

Aqueous solubility:

Slightly soluble

Melting Point:

287.6 °F to 302 °F (142 °C to 150 °C)

Molecular Mass:

399.44

Soluble In:

1 g/220 mL ether; 1 g/100 mL benzene; freely soluble in alcohol or chloroform.

STRYCHNINE

Common Names:

Strychnidin-10-one

Agent Characteristics

APPEARANCE: Colorless, transparent crystals or white, crystalline powder.

DESCRIPTION: Strychnine is a toxic alkaloid derived from the seeds of the trees *Strychnos nux vomica*, *Strychnos ignatii* (S. sancta Ingnatius), and *Strychnos tiente* (Upas tree), that can be found in India, southern Asia, northern Australia, and Hawaii. It was widely used in poison (toxic) baits to kill rodents and other mammals and is a common adulterant of many illicit (street) drugs. Exposure to strychnine can be fatal. It is odorless and has a bitter taste.

METHODS OF DISSEMINATION:

Indoor Air: Strychnine can be released into indoor air as fine particles (aerosol).

Water: Strychnine can be used to contaminate water.

Food: Strychnine can be used to contaminate food.

Outdoor Air: Strychnine can be released into outdoor air as fine particles (aerosol).

Agricultural: If strychnine is released into the air as fine particles (aerosol), it has the potential to contaminate agricultural products.

ROUTES OF EXPOSURE: Strychnine can be absorbed into the body by inhalation or ingestion. It can also be injected into the body when mixed with a liquid.

Personal Protective Equipment.

GENERAL INFORMATION: First Responders should use a NIOSH-certified Chemical, Biological, Radiological, Nuclear (CBRN) Self Contained Breathing Apparatus (SCBA) with a Level A protective suit when entering an area with an unknown contaminant or when entering an area where the concentration of

the contaminant is unknown. Level A protection should be used until monitoring results confirm the contaminant and the concentration of the contaminant.

NOTE: Safe use of protective clothing and equipment requires specific skills developed through training and experience.

LEVEL A: (RED ZONE): Select when the greatest level of skin, respiratory, and eye protection is required. This is the maximum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than the AEGL-2.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A Totally-Encapsulating Chemical Protective (TECP) suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, and a hard hat worn under the TECP suit are optional items.

LEVEL B: (RED ZONE): Select when the highest level of respiratory protection is necessary but a lesser level of skin protection is required. This is the minimum protection for workers in danger of exposure to unknown chemical hazards or levels above the IDLH or greater than AEGL-2. It differs from Level A in that it incorporates a non-encapsulating, splash-protective, chemical-resistant splash suit that provides Level A protection against liquids but is not airtight.

A NIOSH-certified CBRN full-face-piece SCBA operated in a pressure-demand mode or a pressure-demand supplied air hose respirator with an auxiliary escape bottle.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL C: (YELLOW ZONE): Select when the contaminant and concentration of the contaminant are known and the respiratory protection criteria factors for using Air Purifying Respirators (APR) or Powered Air Purifying Respirators (PAPR) are met. This level is appropriate when decontaminating patient/victims.

A NIOSH-certified CBRN tight-fitting APR with a canister-type gas mask or CBRN PAPR for air levels greater than AEGL-2.

A NIOSH-certified CBRN PAPR with a loose-fitting face-piece, hood, or helmet and a filter or a combination organic vapor, acid gas, and particulate car-

tridge/filter combination or a continuous flow respirator for air levels greater than AEGL-1.

A hooded chemical-resistant suit that provides protection against CBRN agents.

Chemical-resistant gloves (outer).

Chemical-resistant gloves (inner).

Chemical-resistant boots with a steel toe and shank.

Escape mask, face shield, coveralls, long underwear, a hard hat worn under the chemical-resistant suit, and chemical-resistant disposable boot-covers worn over the chemical-resistant suit are optional items.

LEVEL D: (GREEN ZONE): Select when the contaminant and concentration of the contaminant are known and the concentration is below the appropriate occupational exposure limit or less than AEGL-1 for the stated duration times.

Limited to coveralls or other work clothes, boots, and gloves.

Emergency Response.

CHEMICAL DANGERS:

Strychnine decomposes on heating, producing toxic fumes, including nitrogen oxides.

Strychnine is incompatible with strong oxidants.

EXPLOSION HAZARDS:

Upper and lower explosive (flammable) limits in air are not available for strychnine.

FIRE FIGHTING INFORMATION:

Strychnine is combustible.

Strychnine is difficult to ignite.

Strychnine decomposes on heating, producing toxic fumes including nitrogen oxides.

For small fires, use dry chemical, carbon dioxide, or water spray.

For large fires, use water spray, fog, or regular foam.

Dike fire control water for later disposal; do not scatter the material.

Use water spray or fog; do not use straight streams.

INITIAL ISOLATION AND PROTECTIVE ACTION DISTANCES:

If a tank, rail car, or tank truck is involved in a fire, isolate it for 0.5 mi (800 m) in all directions; also consider initial evacuation for 0.5 mi (800 m) in all directions.

This agent is not included in the DOT ERG 2004 Table of Initial Isolation and Protective Action Distances.

In the DOT ERG 2004 orange-bordered section of the guidebook, there are public safety recommendations to isolate a strychnine (Guide 151) spill or leak area immediately for at least 150 ft (50 m) for liquids and 75 ft (25 m) for solids in all directions.

PHYSICAL DANGERS:

Not established/determined

NFPA 704 Signal:
Health: 3
Flammability: 1
Reactivity: 0
Special:

Signs/Symptoms

TIME COURSE: Generalized muscle spasms may occur within 5 minutes of inhalation, or intravenous administration, and within 15 minutes of ingestion but may take as long as 60 minutes to appear. Exposure to high levels of strychnine may result in respiratory failure possibly leading to death, and brain death within 15 to 30 minutes following exposure. Seizures may occur within 15 minutes following exposure and generally subside 12 to 24 hours following ingestion. If the patient can be supported for the first 6 to 12 hours, the prognosis is good.

EFFECTS OF SHORT-TERM (LESS THAN 8-HOURS) EXPOSURE: Strychnine is rapidly absorbed after ingestion, inhalation, or intravenous (IV) administration. It causes generalized muscle spasms, muscle cramps, stiffness and tightness, agitation, heightened awareness and responsiveness, respiratory failure, stimulation sensitive seizures, and possibly death.

EYE EXPOSURE:

Not established/determined

INGESTION EXPOSURE:

Possible early adverse effects indicative of strychnine toxicity (prodrome): muscle cramps (especially of the neck and back), stiffness and tightness, agitation, heightened sensory awareness and responsiveness.

Prominent characteristic adverse health effects of strychnine toxicity are painful generalized muscle spasms and seizure. Spasms and seizures are often precipitated by sensory stimulation (sound, touch, and vision). Patient/victims remain alert and lucid during seizures. Seizures may cause hyperthermia, metabolic and respiratory acidosis, destruction of skeletal muscle at the molecular level (rhabdomyolysis), and kidney failure due to the release of muscle protein (myoglobinuric renal failure). Convulsions may be accompanied by abnormal pupil dilation (mydriasis), eye protrusion (ocular proptosis), and rapid uncontrollable eye movements (nystagmus).

Other adverse health effects include fast heart rate (tachycardia), high blood pressure (hypertension), rapid breathing (tachypnea), blood electrolyte and mineral disturbances, an increase of white blood cells (leukocytosis), perspiration (diaphoresis), blue discoloration of the skin due to lack of oxygen (cyano-

sis), jaw closure due to muscle spasms (trismus), facial muscle spasms (risus sardonicus), and spasm of the muscles of the back, causing the head and lower limbs to bend backward and the trunk to arch forward (opisthotonus). Vomiting and nausea occur rarely.

Death may occur due to respiratory failure, cardiac arrest, brain damage, or multi-organ failure.

INHALATION EXPOSURE:

See Ingestion Exposure.

SKIN EXPOSURE:

See Ingestion Exposure.

Decontamination

INTRODUCTION: The purpose of decontamination is to make an individual and/or their equipment safe by physically removing toxic substances quickly and effectively. Care should be taken during decontamination, because absorbed agent can be released from clothing and skin as a gas. Your Incident Commander will provide you with decontaminants specific for the agent released or the agent believed to have been released.

DECONTAMINATION CORRIDOR: The following are recommendations to protect the first responders from the release area:

Position the decontamination corridor upwind and uphill of the hot zone. The warm zone should include two decontamination corridors. One decontamination corridor is used to enter the warm zone and the other for exiting the warm zone into the cold zone. The decontamination zone for exiting should be upwind and uphill from the zone used to enter.

Decontamination area workers should wear appropriate PPE. See the PPE section of this card for detailed information.

A solution of detergent and water (which should have a pH value of at least 8 but should not exceed a pH value of 10.5) should be available for use in decontamination procedures. Soft brushes should be available to remove contamination from the PPE. Labeled, durable 6-mil polyethylene bags should be available for disposal of contaminated PPE.

INDIVIDUAL DECONTAMINATION: The following methods can be used to decontaminate an individual:

Decontamination of First Responder:

Begin washing PPE of the first responder using soap and water solution and a soft brush. Always move in a downward motion (from head to toe). Make sure to get into all areas, especially folds in the clothing. Wash and rinse (using cold or warm water) until the contaminant is thoroughly removed.

Remove PPE by rolling downward (from head to toe) and avoid pulling PPE off over the head. Remove the SCBA after other PPE has been removed.

Place all PPE in labeled durable 6-mil polyethylene bags.

Decontamination of Patient/Victim:

Remove the patient/victim from the contaminated area and into the decontamination corridor.

Remove all clothing (at least down to their undergarments) and place the clothing in a labeled durable 6-mil polyethylene bag.

Thoroughly wash and rinse (using cold or warm water) the contaminated skin of the patient/victim using a soap and water solution. Be careful not to break the patient/victim's skin during the decontamination process, and cover all open wounds.

Cover the patient/victim to prevent shock and loss of body heat.

Move the patient/victim to an area where emergency medical treatment can be provided.

First Aid

GENERAL INFORMATION: Initial treatment is primarily supportive.

ANTIDOTE: There is no antidote for strychnine toxicity.

EYE:

Immediately remove the patient/victim from the source of exposure.

Immediately wash eyes with large amounts of tepid water for at least 15 minutes.

Seek medical attention immediately.

INGESTION:

Immediately remove the patient/victim from the source of exposure.

Ensure that the patient/victim has an unobstructed airway.

Do not induce vomiting (emesis).

Once the airway is secured and neuromuscular activity is controlled, administer charcoal as a slurry (240 mL water/30 g charcoal). Usual dose: 25 to 100 g in adults/adolescents, 25 to 50 g in children (1 to 12 years), and 1 g/kg in infants less than 1 year old.

Treat muscle spasms with diazepam (or other benzodiazepine) under a physician's direction or per local EMS protocol.

If muscle spasms cannot be controlled or recur, administer phenobarbital.

Avoid unnecessary stimulation, which may precipitate additional muscle spasms.

Administer supplemental oxygen and assist ventilation as needed.

For exceptionally high body temperature (hyperthermia), control muscle spasms with diazepam and phenobarbital, remove the patient/victim's clothing, and encourage evaporative heat loss with fans and water to the skin.

Seek medical attention immediately.

INHALATION:

Immediately remove the patient/victim from the source of exposure.

Evaluate respiratory function and pulse.

Ensure that the patient/victim has an unobstructed airway.

If shortness of breath occurs or breathing is difficult (dyspnea), administer oxygen.

Assist ventilation as required. Always use a barrier or bag-valve-mask device.

If breathing has ceased (apnea), provide artificial respiration.

See the Ingestion section for first aid recommendations.

Also see Ingestion Exposure.

Seek medical attention immediately.

SKIN:

Immediately remove the patient/victim from the source of exposure.

See the Decontamination section for patient/victim decontamination procedures.

Seek medical attention immediately.

Long-Term Implications

MEDICAL TREATMENT: If the patient/victim can be rapidly transported to an emergency department following decontamination, stomach pumping (gastric lavage) may be considered after the airway has been secured and neuromuscular hyperactivity is controlled. Gastric lavage is recommended only after ingestion of a life-threatening amount of the agent and only if it can be done shortly after ingestion (generally within 1 hour). Muscle spasms resistant (refractory) to diazepam and phenobarbital plus respiratory failure may require treatment with paralyzing agents, intubation, and mechanical ventilation. Fluid and electrolyte balance should be monitored and restored if abnormal. Measures to prevent and correct accumulation of acid in blood and tissues (metabolic acidosis) should be implemented. Arterial blood gases should be monitored in all symptomatic patient/victims. Dialysis may be necessary in patient/victims with kidney failure.

DELAYED EFFECTS OF EXPOSURE: Not established/determined

EFFECTS OF CHRONIC OR REPEATED EXPOSURE: Information is unavailable about the carcinogenicity, developmental toxicity, or reproductive toxicity from chronic or repeated exposure to strychnine.

On-Site Fatalities.

INCIDENT SITE:

Consult with the Incident Commander regarding the agent dispersed, dissemination method, level of PPE required, location, geographic complications (if any), and the approximate number of remains.

Coordinate responsibilities and prepare to enter the scene as part of the evaluation team along with the FBI HazMat Technician, local law enforcement evidence technician, and other relevant personnel.

Begin tracking remains using waterproof tags.

RECOVERY AND ON-SITE MORGUE:

Wear PPE until all remains are deemed free of contamination.

Establish a preliminary (holding) morgue.

ENVIRONMENT/SPILLAGE DISPOSAL: The following methods can be used to decontaminate the environment/spillage disposal:

Do not touch or walk through the spilled agent if at all possible. However, if you must, personnel should wear the appropriate PPE during environmental decontamination. See the PPE section of this card for detailed information.

Keep combustibles (e.g., wood, paper, and oil) away from the spilled agent. Use water spray to reduce vapors or divert vapor cloud drift. Avoid allowing water runoff to contact the spilled agent.

Do not direct water at the spill or the source of the leak.

Stop the leak if it is possible to do so without risk to personnel, and turn leaking containers so that gas rather than liquid escapes.

Prevent entry into waterways, sewers, basements, or confined areas.

Isolate the area until gas has dispersed.

Ventilate the area.

EQUIPMENT: Agents can seep into the crevices of equipment making it dangerous to handle. The following methods can be used to decontaminate equipment:

Not established/determined

Agent Properties

Chemical Formula:

$C_{21}H_{22}N_2O_2$

Aqueous solubility:

Slightly soluble

Boiling Point:

870 °F (466 °C), decomposes

Density:

Solid: 1.359 at 68 °F/39 °F (20 °C/4 °C)

Flammability:

Combustible

Melting Point:

527 °F to 545 °F (275 °C to 285 °C)

Molecular Mass:

334.41

Soluble In:

Minimally soluble in ether; slightly soluble in acetone, alcohol, benzene, and ethanol; soluble in chloroform.

Учебное издание

Семутенко Константин Михайлович
Ребко Андрей Александрович
Прокопович Дмитрий Александрович

МЕДИЦИНА ЭКСТРЕМАЛЬНЫХ СИТУАЦИЙ
(на английском языке)

Учебно-методическое пособие
для студентов 4 курса факультета по подготовке специалистов
для зарубежных стран медицинских вузов

В двух частях

Часть 2
ТОКСИКОЛОГИЯ ЭКСТРЕМАЛЬНЫХ СИТУАЦИЙ

Редактор ***Т. М. Кожемякина***
Компьютерная верстка ***С. Н. Козлович***

Подписано в печать 03.04.2017.
Формат 60×84$^{1}/_{16}$. Бумага офсетная 80 г/м^{2}. Гарнитура «Таймс».
Усл. печ. л. 11,16. Уч.-изд. л. 12,20. Тираж 50 экз. Заказ № 192.

Издатель и полиграфическое исполнение:
учреждение образования «Гомельский государственный медицинский университет».
Свидетельство о государственной регистрации издателя,
изготовителя, распространителя печатных изданий № 1/46 от 03.10.2013.
Ул. Ланге, 5, 246000, Гомель.